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Aquatic Ecosystem Objectives Committee. Annual Report 1985

Great Lakes Science Advisory Board. Aquatic Ecosystem Objectives Committee

Great Lakes Science Advisory Board
Report to the International Joint Commission

1985 Annual Report

Report of the Aquatic Ecosystem Objectives Committee

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Preface

This report to the Science Advisory Board was prepared by the Aquatic Ecosystem Objectives Committee (AEOC). Though the Board has reviewed and approved this report for publication, some of the specific conclusions and recommendations may not be supported by the Board.

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Summary of Recommendations

The Aquatic Ecosystem Objectives Committee (AEOC) makes the following recommendations to the Science Advisory Board (SAB) for consideration and forwarding to the International Joint Commission (IJC) and the Parties of the Great Lakes Water Quality Agreement (GLWQA).

1. NEW OBJECTIVES

1.1 General Ecosystem Objective (see Chapter 2.1.1)

Article III of the 1978 Agreement should be revised to add: [These waters should be:] . . . (f) maintained and, as necessary, restored to a condition where a balanced and stable community of organisms is present which resembles, as much as is feasible and practicable, the community that existed before the advent of human intervention.

1.2 Oligotrophic Ecosystem in Lake Superior using the Lake Trout as an Indicator of System Health (see Chapter 2.1.2)

Lake Superior should be maintained as a balanced and stable oligotrophic ecosystem with the lake trout as top aquatic predator of a cold-water community.

NOTE: In order to determine whether this condition exists, the following criteria should be met:

(1) the lake trout productivity should be greater than 0.38 kg/ha as determined using mortality rates;
(2) there should be a stable number of recognizable, self-reproducing stocks;
(3) the annual harvest of lake trout should not exceed 0.24 kg/ha;
(4) the harvest of lake trout should be free from contaminants at levels which adversely affect the trout themselves or the quality of the harvested product.

2. REVISED OBJECTIVES

2.1 Ammonia (see Chapter 2.2)

Existing Objective
Concentrations of un-ionized ammonia (NH_3) should not exceed 0.02 milligrams per litre for the protection of aquatic life.
Concentrations of total ammonia should not exceed 0.5 milligrams per litre for the protection of public water supplies.

Recommendation
Concentrations of un-ionized ammonia in water must not exceed 0.03 milligrams per litre for the protection of aquatic life. To protect

aquatic life from short term (<96 hours) exposure to ammonia where limited use zones overlap routes of passage, the estimated lethal threshold concentration on un-ionized ammonia for rainbow trout must not be exceeded. This requirement can be expressed by:

$$\text{mg un-ionized ammonia/L} = 0.3 \times \frac{0.66}{1+10^{(1.03(7.32-\text{pH}))}}$$

2.2 Benzenehexachlorides (see chapter 2.3)

Existing Objective (Lindane)
The concentration of lindane in water should not exceed 0.01 micrograms per litre for the protection of aquatic life. The concentration of lindane in edible portions of fish should not exceed 0.3 micrograms per gram (wet weight basis) for the protection of human consumers of fish.

Recommendation
The concentration of total hexachlorocyclohexane (BHC) isomers should not exceed 0.02 microgram per litre for the protection of aquatic life. The concentration of total BHC isomers in edible portions of fish should not exceed 0.3 microgram per gram for the protection of human consumers of fish.

2.3 Toxaphene (see chapter 2.4)

Existing Objective
The concentration of toxaphene in water should not exceed 0.008 micrograms per litre for the protection of aquatic life.

Recommendation
The concentration of toxaphene in water should not exceed 0.0002 micrograms per litre for the protection of human consumers of fishes.

3. RESEARCH AND OTHER DATA NEEDS

Information needed to permit the development of new objectives or the better establishment of existing ones is listed in Chapter 4. This need takes one of two forms--that which is needed to complete or support an existing, chemical specific objective and that which will identify the expertise which the AEOC feels is necessary to generate such information. These needs are recommended to the Science Advisory Board for their consideration; it is suggested that they be forwarded to the Council of Research Managers.

1. Introduction

During the period of the 1972 Great Lakes Water Quality Agreement, two Committees (Water Quality Objectives Subcommittee -- WQOS and Scientific Basis for Water Quality Criteria Committee -- SBWQCC) were responsible for formulating new or modifying existing water quality objectives. Their collective efforts resulted in Annex 1 of the 1978 Great Lakes Water Quality Agreement. Since the signing of that Agreement, it has been the responsibility of the Aquatic Ecosystem Objectives Committee (AEOC) to ensure that Annex 1 is kept current. In 1980, the AEOC recommended to the Science Advisory Board (SAB) the adoption of two new objectives (pentachlorophenol and polychlorinated dibenzodioxins), the revision of two existing objectives (lead and microbiology) and the adoption of four objectives previously proposed by the WQOS/SBWQCC (silver, chlorine, temperature and nutrients). In 1981, the AEOC's recommendations included the revision of the selenium objective, the confirmation of the mirex objective and the development of a mechanism to define Limited Use Zones. The SAB has concurred with these objectives and the International Joint Commission has recommended them to the Parties, with caveats for chlorine and temperature.

In 1982, the AEOC re-confirmed the silver objective, reviewed a new contaminant (polychlorinated styrenes) and re-examined an old one (asbestos). The AEOC also described a number of research activities required for the development of objectives. In 1983, the AEOC recommended an objective for benzo(a)pyrene and two revised objectives for Microbiology--an indicator species, Escherichia coli and a pathogen, Pseudomonas aeruginosa. Also reviewed was the objective for diazinon and a recommendation for a new level was presented.

In the past year, the AEOC has received the report of the Work Group on Indicators of Ecosystem Quality on the use of the lake trout as an indicator of oligotrophic ecosystem health. Using that document, the AEOC has prepared its own rationale and recommendations for an objective using this species as a measure of the attainment of the objective. The AEOC has also revised the supporting rationales for ammonia, lindane (now incorporated as part of benzenehexachlorides) and toxaphene and has made new recommendations based on these evaluations.

The framework[1] for developing objectives was formulated by the WQOS/SBWQCC and is restated here for the sake of clarity. It is understood that the terms water quality and ecosystem quality are equivalent for the purposes of the AEOC.

1. In developing specific water quality objectives, the philosophy of protecting the most sensitive use is employed.

2. The objectives serve as a minimum target wherever water quality objectives currently are not being met.

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[1] International Joint Commission. New and Revised Great Lakes Water Quality Objectives. Volume II. Washington, D.C. and Ottawa, Ontario, October, 1977. pp. 3-7.

3. For jurisdictionally-designated areas that have outstanding natural resource value and existing water quality better than the objectives, the existing water quality should be maintained or enhanced.

4. Specific water quality objectives are to be met at the periphery of mixing zones. This assumes that water quality conditions better than the objectives will result beyond the mixing zones. The objectives should be implemented in concert with limitations on the extent of mixing zones or zones of influence and localized areas as designated by the regulatory agencies.

5. In recommending objectives to protect raw drinking water supplies, it has been assumed that a minimum level of treatment is provided before distribution to the public for consumption.

6. Adoption of objectives does not preclude the need for further study of the effects of pollutants on the aquatic environment.

7. Since infinite combinations of water quality characteristics may occur, the objectives often are unable to take into account antagonistic, synergistic and additive effects because of lack of data.

8. Since new data may lead to modified recommendations, the objectives are subject to continual review.

9. No adequate scientific data base can exist for establishing scientifically justifiable numerical objectives for "unspecified non-persistent toxic substances and complex wastes". Therefore, criteria for developing an operationally defined objective for local situations have been recommended.

The AEOC endorses this framework with the understanding that objectives do not consider socio-economic factors. The committee agrees with previous recommendations (Water Quality Board, 1980)[2] that socio-economic impact assessment is the responsibility of the jurisdictions and should be done at the time of setting of regulations or standards. Objectives should not be construed as regulations or standards but should be considered as goals to be achieved and as a minimum basis for developing regulations or standards by the jurisdictions.

In the course of their development, the objectives have been subject to iterative reviews within the committee and by scientists with relevant expertise. The committee, however, welcomes any comments or additional scientific evidence relevant to any of the objectives and consistent with the above philosophy.

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[2] Alternatives for Managing Chlorine Residuals: A Social and Economic Assessment. Final Report of the Chlorine Objectives Task Force to the Great Lakes Water Quality Board. Windsor, Ontario, April, 1980.

2. Objectives

2.1. ECOSYSTEM

2.1.1 General Objective for Ecosystem Quality

Recommendation

Article III of the 1978 Agreement should be revised by adding: [These waters should be:] .....(f) maintained and, as necessary, restored to a condition where a balanced and stable community of organisms is present which resembles, as much as is feasible and practicable, the community that existed before the advent of human intervention.

Rationale

The United States and Canada signed a revised and strengthened Great Lakes Water Quality Agreement in 1978. At that time, certain weaknesses in the traditional 'chemical-by-chemical' water quality objectives approach were recognized and great emphasis was placed on using in situ biological components of the ecosystem to evaluate the biological integrity of the waters of the entire Great Lakes Basin.

To further the goal of protecting the biological as well as the chemical and physical integrity of the system, the International Joint Commission's (IJC) Science Advisory Board (SAB) established the Aquatic Ecosystem Objectives Committee (AEOC) and charged them with the task of developing the aquatic ecosystem objectives called for under the Agreement. These have traditionally been of the sort that limit the concentrations of chemicals in the waters or tissues of fishes to levels which scientific evidence indicates should be "safe"; they have not been "objectives" in the sense of goals to be sought or achieved.

The AEOC concluded that the spirit of the Agreement could also be met by setting a General Objective for the restoration and maintenance of Great Lakes biological communities similar to those present before the modification by human intervention. Such an objective has already been recommended to the SAB for transmittal to the IJC and the Parties and is re-presented here in the AEOC's Annual Report. This General Objective complements, the AEOC feels, the Specific Objective for Lake Superior which forms the next sub-chapter in this report. The purpose of this General Objective is to provide a philosophical framework for the development of Specific Ecosystem Objectives. It basically calls for the maintenance or restoration, to the extent possible, of conditions that the Great Lakes are known to be capable of sustaining.

2.1.2 Objective for a Healthy Lake Superior Ecosystem (Lake Trout Indicator)

Recommendation

Lake Superior should be maintained as a balanced and stable oligotrophic ecosystem with the lake trout as top aquatic predator of a cold-water community.

NOTE: In order to determine whether this condition exists, the following criteria should be met:

(1) the lake trout productivity should be greater than 0.38 kg/ha as determined using mortality rates;

(2) there should be a stable number of recognizable, self-reproducing stocks;

(3) the annual harvest of lake trout should not exceed 0.24 kg/ha;

(4) the harvest of lake trout should be free from contaminants at levels which adversely affect the trout themselves or the quality of the harvested product.

Rationale

Introduction

In fulfillment of its mandate, the Aquatic Ecosystem Objectives Committee (AEOC) determined that its efforts to develop Ecosystem Objectives would take on a new approach in addition to the continued description of upper limits to specific chemical concentrations in various parts of the Great Lakes. The new approach is intended to provide true ecosystem "objectives" in the sense that they describe a condition to be sought for all parts of the system. Such objectives are intended to include consideration of community properties and behaviour; deviations from the objective would indicate the existence of an ecosystem stress, the precise nature of which might need to be determined for remedial action to be taken.

In order to implement the spirit of the General Objective (see chapter 2.1.1), the Specific Objective has been developed for the Lake Superior ecosystem. While the boundaries and means of measuring the health of ecosystems are relatively untried, it was felt that in the case of this particular system, the data base was adequate to permit such a development. The AEOC considered that they could specify the relative abundance or condition of key biological indicators which would be representative of the overall condition or health of the ecosystem. A study with this approach was

undertaken by a Work Group which produced the report--"A Conceptual Approach for the Application of Biological Indicators for the Determination of Ecosystem Quality in the Great Lakes" (Ryder and Edwards, 1985). This evaluation concluded that for Lake Superior, the lake trout (Salvelinus namaycush) was an appropriate organism as it integrates, through terminal predation, much of the biological structure of that large, oligotrophic lake system. The balance of this rationale deals with the measurement and justification of several aspects of the lake trout as such an indicator. Since Lake Superior is a sub-component of the Great Lakes for which a large body of lake trout information exists and because it is the Great Lake least affected by humans, the objective has been written with this lake in mind. The principles, however, apply to all lakes as well as to their biotic communities. The reader is referred to the above report for additional material and references.

Criteria for an Ecosystem Indicator

An ecosystem may be perceived as having many hierarchic levels (Allen and Starr, 1982) that are closely interwoven in the biotic sector through various interdependencies such as food-web interactions and in the abiotic sector, through water exchange, nutrient leaching and energy flows, among other factors. The biotic and abiotic sectors, in turn, interact at organism-environment interfaces where exchanges of materials and energy can occur in either direction. It is impossible to monitor all species and their related biological and abiotic parameters; it may be possible, however, to assess the health of an ecosystem by monitoring a small number of key organisms. With such an approach, a single organism or suite of organisms is employed as a surrogate for the whole community of organisms of which it is a vital component. Criteria for such an "umbrella" organism dictate a number of requisite attributes that must be met before an adequate representation of the state-of-health of its constituent community may be made. Generally, a good candidate will meet many of these, but not necessarily all. The selected indicator will:

- have a broad distribution in the system;
- be easily collected and measured in terms of biomass;
- be indigenous and maintain itself through natural reproduction;

- interact directly with many components of its ecosystem;
- have historical, preferably quantified, information pertaining to its abundance and other critical factors relevant to the state of the organism;
- have well documented and quantified niche dimensions expressed in terms of metabolic and behavioural responses;
- exhibit a gradual response to a variety of human induced stresses;
- serve as a diagnostic tool for specific stresses of many sorts;
- respond to stresses in a manner that is both identifiable and quantifiable;
- be a suitable species for laboratory investigations;
- be generally recognized as important to humans; and
- serve to indicate aspects of ecosystem quality other than those represented by presently accepted parameters.

The importance of these requirements are, for the most part, obvious. Indicator species are intended to reflect the natural system and hence the requirements for natural reproduction, abundance and indigenous distribution. An indicator is also intended, as its primary purpose, to reflect stresses and to the extent possible, to help in their identification. The remaining requirements are primarily practical and are important for that reason.

A variety of organisms was considered for use as indicators of environmental quality in cold-water oligotrophic ecosystems. The lake trout was judged to be the one that came closest to meeting the criteria listed above for oligotrophic areas of the Great Lakes, specifically for Lake Superior. The following points indicate the ways in which this species meets these criteria:

- lake trout, in the early days of settlement, occupied almost every major habitat type, at least at certain times of the year. This included the pelagic, demersal and near-shore littoral zones as well as the lower reaches of major tributary streams (Lawrie, 1978);

- harvest of lake trout is easily sampled through existing commercial fisheries, through creel census of sports fisheries or through purchases from wholesalers or the retail market;

- although reduced in numbers from historical times, the lake trout have maintained sizeable, naturally reproducing populations in most parts of Lake Superior;

- they have been a structurally stable component of a relatively unperturbed, oligotrophic system in Lake Superior over a long enough period of time for evolution to occur (Bailey and Smith, 1981);

- as the top predator, the lake trout interacts with a large variety of indigenous organisms through its place as the top predator in the food web and through competition with other coldwater predators and in early life stages, with planktivores (Martin and Olver, 1980; Ryder, et al., 1981);

- historical data stretching back into the 1800's exist on the abundance of the lake trout in each of the Great Lakes as determined from fishery harvests (Baldwin, et al., 1979). Prior to this, there are personal accounts that describe lake trout stocks (Agassiz, 1850) and their relative abundance (Smith, 1976);

- the niche characteristics of the lake trout and its habitat requirements, including limits and preferred conditions of environmental properties such as temperature and dissolved oxygen, are well-established;

- the lake trout exhibits a gradual response with respect to environmental stresses such as cultural eutrophication (Loftus and Regier, 1972), acid precipitation (Kennedy, 1980), loadings of toxic materials (Willford, et al., 1981), exploitation, implantation or invasion by exotic species (Pycha, 1980), water level controls (Martin, 1955) and a variety of physical habitat alterations;

- while the lake trout is not commonly used as a laboratory test species, the procedures for maintaining all of its life stages are well-established and it has been occasionally used in this manner (Eaton, et al., 1978; Passino and Coutant, 1979).

- the lake trout at one time provided one of the largest and most valuable commercial fisheries in the Great Lakes (Baldwin, et al., 1979). It is still a major sport fish species in Lake Superior and generates large incomes from

tourist activities. It is generally recognized by the public as a desirable species with aesthetic qualities which they believe to be indicative of a healthy environment;

- because the lake trout is a major terminal predator in Lake Superior, aspects of ecosystem quality determined through its study are integrative and necessarily incorporate aspects of other system components, biotic and abiotic, at other trophic levels.

A healthy coldwater, oligotrophic ecosystem is generally held to be both the historic and present day condition appropriate to most portions of the upper Great Lakes and to Lake Superior in particular. Accordingly, the lake trout was selected as an objective for Lake Superior. It is believed by the AEOC that an objective based upon the well-being of lake trout stocks would most satisfactorily reflect the relative condition of the whole biotic community. Any cultural intrusions, be they contaminant or nutrient loadings, structural changes to the waterways, increased turbidity and sedimentation, overfishing, incursions of sea lampreys or other exotic species, would be mirrored in the relative condition of the lake trout stocks as compared with those in the 1800's and the first half of this century (Loftus and Regier, 1972). Any marked deviation from normal variability would indicate an innate system malaise not apparent to the casual observer. Precise identification of the cause of the stress would not necessarily be indicated at first examination, but the knowledge of its presence would be an advantage in later identification.

The Lake Trout in Lake Superior

Early evidence circa 1800, indicates that Great Lakes fish communities of that time differed substantially from those encountered today (Loftus and Regier, 1972). Typical of the differences in the fish communities of that time was the presence of a relatively high abundance of large littoral and demersal terminal predators such as lake trout and burbot (Lota Lota Linnaeus) and benthic feeders such as the lake sturgeon (Acipenser fulvescens Rafinesque) as well as river-run species that now occur only in deep-spawning forms (Smith, 1972a). Mean sizes of most species were markedly larger in the near-pristine environment of the time and mean ages of mature fishes in spawning runs were substantially

greater (Loftus and Regier, 1972) than at present. High levels of morphological and functional diversity existed in lake trout and cisco stocks and allowed for ready adaptation to the variety of habitats in the lake basins.

Cultural stresses around 1800 were relatively unimportant in affecting fish abundance except in certain local instances (Loftus and Regier, 1972; Smith 1976). The implanted and invading species of ecological and economic importance today were not yet present in the four upper lakes. The sea lamprey and alewife may have made some early incursions into Lake Ontario but even there, the full impact of these two species was not fully felt until deforestation was sufficiently widespread to affect adversely the mean water temperatures of influent streams (Smith, 1976). Soil regimes were also adversely affected by deforestation and the large zones of sedimented organic materials that developed in some streams may have increased the habitat for ammocoetes (larval sea lamprey) (Smith, 1976). Svardson (1975) has suggested that a pervasive, environmental stress is necessary before the impact of an exotic species can be felt. As in Lake Ontario where other factors contributed to the impact of the sea lamprey on lake trout, such stresses may also have contributed to the negative effects that this species had on lake trout in other parts of the Great Lakes.

Historically, the species of prime importance from both an ecological and economic point of view in the upper Great Lakes was the lake trout. This terminal predator occupied most of the lake basin, preying upon a spectrum of food organisms, but principally the whitefish (Coregonus clupeaformis Mitchill), sculpins, crayfish, water fleas and scuds (Eschmeyer, 1964). The burbot was the only other top predator that occupied the cold, deep waters of the region and this species was at least partially isolated from direct competition with the lake trout since it is more of a benthic scavenger and is not found at such depths as are utilized by many of the lake trout stocks (Scott and Crossman, 1973). A large coregonine swarm of perhaps eleven species of chubs, ciscoes and whitefish existed in Lake Michigan and somewhat fewer species in Lakes Huron and Superior (Koelz, 1929) where they constituted the bulk of the remaining fish stocks forming the cold-water community.

Today, as a generalization, the fish communities of the Great Lakes Basin including the lake trout, may be described as of smaller mean size, comprising species more dependent on the pelagic zone and lacking large specimens that were formerly abundant in rivers and near-shore zones. Additionally, these communities are often dominated by opportunistic invaders such as the sea lamprey, alewife and rainbow smelt, or by exotic

species such as the carp and implanted Pacific salmons. Available evidence suggests present fish community is less stable and diverse than that which existed in the Great lakes two hundred years ago (Ryder and Kerr, 1978; Ryder, et al., 1981). The composition of this present-day community is constantly changing--moving further away from its natural state in response to continued cultural stresses that are imposed on it.

Complementary to the genetically determined limitations of niche boundaries, there are those imposed by the habitat itself. Habitat constraints, at their most fundamental level, are reflected as inputs of materials and energy. Nutrients and oxygen are two key material components commonly identified as essential to community survival; temperature and light represent energetic inputs. The limits of these inputs have been well-defined for different life stages of the lake trout, but are better quantified for adults (Martin and Olver, 1980).

Light and nutrients, while having a strong influence on fish communities over diurnal and annual time periods, are rarely lethal to fishes per se although they may induce marked behavioural changes (Ryder, 1977). Light on a diurnal scale affects feeding and on an annual scale, establishes the start of spawning; temperature regime regulates the duration of the latter. Optimum temperature for growth and other metabolic activity of the lake trout is about 10.5°C (Coutant, 1977). Minimal oxygen levels for assured survival in the summer hypolimnion is at least 5.5 mg/L (Magnuson, et al., 1979) although Martin and Olver (1976) suggest a lower value. Nutrient levels for optimal lake trout growth and reproduction are about 10 mg phosphorus/L. Such levels, however, may result in increased primary productivity with resulting oxygen deficits in thermally stratified parts of oligotrophic lakes. Sedimentation of organic material on spawning grounds under these conditions may be sufficient to cause an increased oxygen demand and thereby restrict reproduction. In Lake Superior, none of these conditions is expected to become limiting.

Photoperiod, temperature, oxygen and nutrients are also important regulators of primary producers, which convert energy inputs into usable forms for the ecosystem in general. Therefore, while these inputs directly characterize the physical niche dimensions, they also indirectly control the biotic dimensions for lake trout growth and reproduction.

Dramatic changes may occur in the biota at all trophic levels of streams and river coincident with declining pH (Wright and Snekvik, 1978; Harvey, 1980; Cowling and Linthurst, 1981). The loss of lake trout populations in aquatic ecosystems experiencing a decline in pH is well-documented (Jensen and

Snekvik, 1972; Beamish, 1974, 1975; Scholfield, 1975). Generally, lake trout populations may show measurable stress at pHs of about 6.0 (Kennedy, 1980); they cease reproduction at about pH 5.5 and become extinct at values of pH below 5.0. The pH of very soft water in streams entering Lake Superior might be reduced by cultural acidification to levels harmful to lake trout spawning there (Loftus, 1958). This is not expected to be a problem for the lake spawning stocks, however.

Lake Trout Stock Diversity

A unique characteristic of the early lake trout and coregonine stocks of the upper lakes was their diversity of form and behaviour (Goodier, 1981; Todd, 1981). Because of an overlap in morphological attributes among the various forms within lake trout and ciscoe populations, their precise taxonomic classification remains open to question (Bailey and Smith, 1981). As an example, the fact that cross-breeding appears to take place easily and frequently among the Lake Superior ciscoe stocks, suggests that these forms should rank only as phenotypic stocks rather than species (Todd, 1981). Of critical importance, however, is the fact that each taxon flock functioned as a separate species, efficiently exploiting otherwise unoccupied habitats through rapid phenotypic adaptation (Ryder, et al., 1981) presumably assisted by reproductive isolation.

Each spawning area had its morphologically and biologically distinctive stocks (Brown, et al., 1981; Goodier, 1981). Some stocks appeared to spend their entire lives in the vicinity of their spawning area while others ventured across the entire lake (Rahrer, 1968). All apparently exhibited a moderately strong homing instinct to their natal spawning area as they matured (Martin, 1960), a property that reduced gene flow among the different spawning aggregations and contributed to the genetic distinctness of the stocks. At least 200 former spawning grounds have been identified in Canadian waters including 20 rivers characterized by September spawning runs of lake trout (Goodier, 1981). Other stocks are reported to spawn from early summer until well into the winter (Eschmeyer, 1956). Given a sufficiently long evolutionary time and continued reproductive isolation, the phenotypically recognizable entities may eventually have speciated and a genetic drift may have resulted in an even closer adaptation to particular habitats (Fryer and Iles, 1972).

Of the recognizably different stocks of lake trout to be found in the Great Lakes about a century ago, only a few have survived (Spangler, et al., 1981). This is also the case for some of the coregonine stocks where inter-stock introgression

has reduced their diversity. Current attempts to restore the lake trout to something akin to its earlier status involve efforts to create a genetically more diverse stock structure (Schneider, et al., 1983). Resource management to date has largely been a matter of trial and error but the experience of the 1980 Stock Concept International Symposium (STOCS) (Berst and Simon, 1981) encourages a more informed approach that maximizes genetic variability while avoiding undesirable changes (Hynes, et al., 1981; Krueger, et al., 1981).

Lake Trout Reproduction and Growth

Prior to 1900, various stocks of lake trout were present in Lake Superior, some spawning primarily over large rubble or boulder substrates at depths of less than 35 metres while others spawned in connecting waters and certain tributaries of the lake (Loftus, 1958). At present, spawning of the reduced stock generally occurs in autumn at which time 5-6 mm eggs (200-600/kg of female) are distributed over rocky substrates and hatching occurs in 15-21 weeks at 0.3-1° C. Survival of sac-fry, yearlings and juveniles of natural stocks has not been adequately described, as young fish seek deeper waters within a month of yolk sac absorption. Annual adult mortality figures for western Lake Superior of 32-85% (Swanson and Swedberg, 1980) and 50-60% for eastern Lake Superior (Pycha, 1980), often exceed the maximum tolerable annual mortality rate of 50% for a naturally reproducing lake trout population (Healey, 1978). Growth rates for the 0+ to 10+ year class fish based upon inland population data for appropriate large lakes, suggest that 6.4 (S.D. ± 0.5) cm/annum would appear normal (Scott and Crossman, 1973). Fish become sexually mature at age 6-7 years and can continue to grow, exceeding 20 kg. Figure 1 presents data for Lake Superior lake trout; the resulting correlation indicates a growth rate of 7.2 (S.D. ± 0.2) cm/year (Rahrer, 1967).

Stresses that directly increase mortality rates may have the effect of reducing intra-stock competition for food and thus lead initially to increased growth rates (e.g. Ryan and Harvey, 1977). However, chronic stresses that lead to reduced viability over extended periods, such as increases in disease, parasitism and contaminant loading, will reduce growth rates. Such factors should therefore be considered if the observed growth rate is markedly different from the 7.2 cm/year expected.

Sea Lamprey Predation on Lake Trout

Of the many exotic species of fishes introduced into or invading the Great Lakes, only the sea lamprey (Petromyzon marinus) had effects so severe that the lake trout ultimately became extinct in some lakes. Present day control of the sea lamprey (Smith, 1972b; Smith, 1973) has succeeded in reducing its abundance to 10-20% or less of its peak. This will likely result in the survival of the lake trout, certainly in the upper Great Lakes, but the stress (or threat of it) will continue for the foreseeable future. Wounding and scarring rates on the lake trout due to this predation should therefore be monitored on a continuing basis (Pycha, 1980). Fresh wounding, such as is found in the spring, can be used to estimate the mortality due to the sea lamprey. A 2% wounding rate at this time in specimens over 53 cm reflects a 5% induced mortality; smaller fish are markedly less able to survive these attacks. The 2% rate is conservative and would provide protection, both for the mature fish as well as the young. Sea lamprey induced mortality, as estimated from such wounding rates, must be compensated for by reduced harvests in order to limit total mortality to acceptable levels.

Lake Trout Productivity and Harvest in Lake Superior

The historical lake trout stocks exhibited a high degree of adaptation to a wide variety of ecological zones in the lakes. Intensive fishing effort during the period 1900-1950 depleted certain stocks, while maintaining a constant overall yield by successfully fishing-up other stocks. Those stocks that were the most accessible and commercially desirable were usually depleted first while less desirable stocks were influenced little, if at all, by this exploitation (Brown, et al., 1981). Young lake trout of certain stocks occupied the relatively shallow region close to the spawning areas and moved to deeper waters as they matured (Eschmeyer, 1956). Adults of some races remained primarily bottom dwellers and therefore were more vulnerable to exploitation; others were mostly pelagic, except at spawning, and were virtually immune. These latter stocks became increasingly vulnerable as the benthic stocks were sequentially depleted. Some of those, from distant shoals such as the Superior shoals, have been little influenced by such fishing in the past because of their remoteness and because they escaped the full brunt of the sea lamprey, which does not usually venture offshore (Rahrer, 1965). These stocks form some of the brood material for the rehabilitation efforts of today.

The maximum sustainable harvest of lake trout from Lake Superior can be estimated from an examination of historical yields summarized from Baldwin et al. (1979) in Table 2.1.1.

Table 2.1.1
Annual Fish Harvest in Lake Superior (millions of kg)

YEARS	LAKE TROUT	ALL SPECIES
1879-1900	1.97	3.94
1901-1920	2.04	6.90
1921-1930	1.98	7.22
1931-1940	2.00	8.57
1941-1950	2.07	9.71
1951-1960	1.17	7.72
1961-1970	0.14	5.39
1971-1977	0.21	4.12

Records for each annual harvest exist from 1867 onward for the Canadian catch but U.S. data are sporadic until 1913. The Canadian harvest ranged from 0.036 to 0.31 million kilograms from 1867 to 1880, increased during the 1880's and was consistently above 0.45 million kilograms from 1891 to 1955 (except for 1933 at 0.44 million kilograms). U.S. harvests after 1913 were similarly steady with yields consistently in the 0.91 million kilogram range. Total harvests of lake trout remained high until 1950 and no marked changes in the mean annual harvest occurred from decade-to-decade during the first half of the century. Harvests dropped sharply in the 1950's and have been low ever since. The mean annual harvest from 1901 to 1950 (n=40 excluding 10 years with incomplete data) was 2.0 million kilograms (95% confidence limits 1.9-2.1), equivalent to 0.24 kg/ha. The long-term stable harvests and eventual decline suggest that annual harvests can approach, but should not exceed this figure in order to ensure a healthy lake trout population.

A healthy ecosystem will have substantial production of top predators, whether these are harvested or not. The level of production of lake trout in Lake Superior can be estimated from mortality rates. Maximum sustainable mortality of this species is reported to be 50% per annum (Healey, 1978)--equivalent to an instantaneous mortality rate of 0.69/year. Instantaneous natural mortality of the lake trout in Lake Superior has been estimated to be 0.26/year (Pycha, 1980). Assuming that total mortality was close to the maximum sustainable by the lake trout during the first half of the 20th century, the instantaneous mortality due to fishing would have been 0.69-0.26 = 0.43/year and lake trout total production would therefore be 0.24 kg/ha x 0.69/0.43 = 0.38 kg/ha per annum (or greater since not all lake trout size classes are fished). Production of top predators in Lake Superior should therefore be at least at this level in a healthy

ecosystem. This production can be supported by a large biomass of fish with a low turnover rate if not harvested (e.g. 0.38/0.26 = 1.46 kg/ha standing stock with an instantaneous mortality or turnover of 0.26/year assuming no mortality due to lamprey predation), or alternatively, by a smaller biomass with a higher turnover rate if harvested (e.g. 0.38/0.69 = 0.55 kg/ha standing stock with a total instantaneous mortality of 0.69/year including natural mortality, fishing and lamprey predation).

An adequate food source for the lake trout must exist if historical production levels are to be achieved. Lake Herring (Coregonus artedii Lesuer)and other ciscoes, were a major food source of the lake trout in the early 1950's (Dryer, et al., 1965). During the first half of the 1900's, the mean annual harvest of lake herring was 5.2 million kilograms (0.63 kg/ha), or 64% of the total Lake Superior fish harvest for all species (Baldwin, et al., 1979). Most total annual harvests were near 3.6 to 5.5 million kilograms in the early 1900's, followed by harvests near 5.9 to 8.2 million kilograms in the 1940's. These latter were followed in turn by decreasing harvests and the demise of the fishery suggested that this harvest rate was too high. Maximum sustainable forage fish harvests should therefore probably be no more than 0.5 kg/ha to ensure a continuous food supply for the lake trout.

One feature not found in the harvest statistics but of considerable importance in predicting future harvests, is the contribution of different stocks to the total lake trout harvest. The constant harvest prior to 1950 is believed to have been maintained largely by switching from one stock to another as each in turn became scarce. It also appears that the constant harvest was partially maintained through an increased fishing effort, with lake trout abundance actually decreasing from 1930 onward (Lawrie and Rahrer, 1973). Consequently, the constant lake trout harvests noted above do not necesarily represent a sustainable yield for the lake as a whole. Perhaps this level of production can be supported if all stocks are rehabilitated and subsequently fished simultaneously, each at its own appropriate level. Sequential fishing of one stock after another will lead to extinction as it has in the past. Proper stock management is therefore a prerequisite to attaining sustained future lake trout harvests in Lake Superior.

Contaminants in Lake Trout

The reproductive and early life history stages of the lake trout are especially vulnerable to environmental stresses including those imposed by contaminants. Recruitment to a stock can easily fail if the eggs carry a sufficiently high burden of contaminants (Burdick, et al., 1972) so that the fry are poisoned when the egg material is metabolized. Willford, et al., (1981) demonstrated such an impact on lake trout from Lake Michigan over the period 1972 to 1977.

Lake trout, like other species, can be expected to demonstrate other symptons of contaminant stress in addition to those associated with their reproductive and early life stages. Changes in activity and skeletal deformaties have been noted following exposure to chemicals such as dieldrin, endosulfan, 2,4-D, PCB's, malathion, carbaryl, atrazine and chlordane (Meyers and Hendricks, 1982). Sublethal metabolic or behavioural changes have also been observed in Atlantic salmon (Anderson, 1971; Anderson and Peterson, 1969; Anderson and Prins, 1970; Ogilvie and Anderson, 1965). Other effects include external tumors and other anomalies in fishes (Black, 1983; Baumann, 1984); necrotic tissue and degenerative organ function, including testicular, in rainbow trout (Meyers and Hendricks, 1982). These effects are those from exposure to xenobiotic substances observed in the field; some of them have also been demonstrated in the laboratory.

Metals of concern include cadmium, copper, mercury and lead--all of which have impacts on multiple generations of fishes (McKim and Benoit, 1971; Benoit, et al., 1976; Holcombe, et al., 1976). These metals have been shown to be lethal to lake trout and other salmonids at concentrations only somewhat above those observed in parts of Lake Superior. Cadmium has been shown to kill embryonic and larval lake trout and to produce a reduction in growth rate of the survivors (Eaton, et al., 1978); lead exposure is known to result in blacktail, a symptom of neurotoxicity which is determined by the growth rate of the fish (Hodson, et al., 1982). Mercury at concentrations below the detection level may cause concern through accumulation to toxicologically significant levels in lake trout tissues (Moore and Sutherland, 1980). Although mercury is generally found in the inorganic form in the lakes themselves, it is the more toxic methyl mercury that it is found in fish tissues (Biesinger, et al., 1982; Mason, 1981).

Anthropogenic contamination of lake trout populations in Lake Superior is primarily in the form of metals and persistent organic chemicals. The lake trout is an efficient accumulator of many of these and in cases, where the data base for human consumption has warranted it, the sale and/or consumption of the lake trout has been restricted (e.g. MOE, 1984). A number of objectives for these chemicals in fish tissues have been developed under the Great Lakes Water Quality Agreement. These are to be found in reports of the Water Quality Objectives Sub-committee/Scientific Basis for Water Quality Criteria Committee (WQOS/SBWQCC, 1974 and 1975) and in reports of the AEOC (1980, 1981, 1983 and 1985). All should be met or bettered in order to provide protection to the fish and acceptability to the consumer.

Dichotomous Key Program for Evaluating Lake Trout Data

Any objective, whether General or Specific, requires feedback to determine progess towards meeting its desired goal. In the case of the traditional Specific Objective, definitive tests can determine quantitatively whether it is being met. A holistic objective (such as one for a "healthy Lake Superior ecosystem") cannot have such a direct means of measuring progress and can only be estimated indirectly. An indicator or indicators (e.g. the lake trout) must be employed as a surrogate which will indicate whether the objective is being met.

The information on the lake trout that is usually available to the water or fishery manager, has been utilized in a computer program referred to as the "Lake Trout Dichotomous Key". This program, designed for use with a micro-computer, can be found in Ryder and Edwards (1985) as Appendix VI. It asks questions under five main headings: (1) exploitation; (2) sea lamprey; (3) contaminants; (4) environment (biotic); and (5) environment (abiotic). Specific questions are asked requiring a "Yes" or "No" response from the answers, an indication of the progress towards meeting the objective is given. This takes the form of a statement that the system is stressed (and what the possible stresses are), or, that the system is not stressed. Illustrative of the questions asked are:

- is the total annual mortality rate (fishing plus natural) for lake trout less than 0.5/year (i.e., 50%)?

- are all the current water quality objectives (Annex 1) for [levels of contaminants] being met?

- is the proportion of yearling lake trout of native or wild origin greater than 90%?

- do spring catches of lake trout >53 cm in length exhibit a lamprey wounding rate of <2% (unhealed wounds)?

- is the spawning substrate large enough (2-20 cm) and deep enough (>15 cm) to permit infiltration of lake trout eggs into interstitial spaces which are small enough to prevent penetration by predators?

The computer program is designed to use the lake trout as the indicator but similar keys using other appropriate indicator species can be designed to assess water bodies other than those of cold, oligotrophic lakes.

Summary

The general conclusion is drawn from the preceding is that an oligotrophic, coldwater ecosystem is the most appropriate state for Lake Superior and that its condition in this regard can best be determined from an examination of the state of the lake trout.

The following recommended criteria should be evaluated on an annual basis:

(1) The lake trout productivity should be greater than 0.38 kg/ha as determined using mortality rates;

(2) there should be a stable number of recognizable, self-reproducing stocks;

(3) the annual harvest of lake trout should not exceed 0.24 kg/ha; and

(4) the harvest of lake trout should be free from contaminants at levels which adversely affect the trout themselves or the quality of the harvested product.

Several aspects of the life cycle of the lake trout have been identified in the preceding sections. Failure to reach certain levels for some of these criteria may prove limiting to the healthy condition of this species in Lake Superior. Particular among these criteria which the AEOC feels are important both to the lake trout and its acceptability is its contaminant load. Other criteria are also influential in achieving these goals but it is felt that knowledge of these four will permit a reasonable assessment of the quality of the ecosystem. If the criteria are not met, then as a protective measure, the system must be assumed not to be healthy and the use of the Dichotomous Key program will be of assistance in diagnosing the nature of the stresses.

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2.2 AMMONIA

EXISTING OBJECTIVE

Concentrations of un-ionized ammonia (NH_3) should not exceed 0.02 milligrams per litre for the protection of aquatic life. Concentrations of total ammonia should not exceed 0.5 milligrams per litre for the protection of public water supplies.

RECOMMENDED OBJECTIVE

Concentrations of un-ionized ammonia in water must not exceed 0.03 milligrams per litre for the protection of aquatic life. To protect aquatic life from short term (<96 hours) exposure to ammonia where limited use zones overlap routes of passage, the estimated lethal threshold concentration on un-ionized ammonia for rainbow trout must not be exceeded. This requirement can be expressed by:

$$\text{mg un-ionized ammonia/L} = 0.3 \times \frac{0.66}{1+10^{(1.03(7.32-pH))}}$$

RATIONALE

Introduction

Ammonia continues to be a major water pollutant with a potential for producing serious detrimental effects on aquatic organisms. Ammonia enters surface waters directly via urban, industrial and agricultural discharges and to a lesser extent through precipitation and gas exchange with the atmosphere.

Indirect sources include the chemical and biochemical transformation of nitrogenous organic and inorganic matter in soil and water. Some of these processes oxidize ammonia to nitrite and nitrate ions, while others incorporate dissolved nitrogen gas into organic matter, through the formation of proteins. Another source, although of limited significance, is the excretion of ammonia by biota.

The previous objective (IJC, 1974) was developed by the multiplication of a 0.05 safety factor by the lowest acute lethal concentration of 0.5 mg/L un-ionized ammonia for rainbow trout (Salmo gairdneri) and 0.28 mg/L for Atlantic salmon (Salmo salar). The objective of 0.02 mg/L represented a compromise between the two limits (0.025 mg/L and 0.014 mg/L) calculated above and was considered to approximate closely the observed sub-lethal threshold for rainbow trout growth reported by Smith and Piper (1975). The effect of water quality on the potency of un-ionized ammonia was not considered previously as there was insufficient indication that such interactions were significant. Water temperature and pH were considered important only in the calculation of the degree of ammonia ionization.

A number of investigators have recently described the modifying effect of pH on un-ionized ammonia toxicity so that a larger data base is currently available for comparison of predicted toxic levels. The sublethal toxicity data base is also more extensive and provides the opportunity to develop an objective based directly on chronic toxicity.

This revised objective also provides short term protection for fish passing through limited use zones by accounting for the increased toxicity of un-ionized ammonia under low pH conditions. Application of this objective will be significant in providing equivalent water quality protection in tributaries and nearshore areas of the Great Lakes where receiving water pH differs geographically or seasonally. Furthermore, this part of the objective is developed from a more extensive data base than existed previously and is to be met within the appropriate limited use zone.

Chemical-Physical Equilibria of Ammonia

Environmental ammonia equilibrates between the ionized (NH_4^+) and un-ionized (NH_3) form, with the latter being about two orders of magnitude more toxic for fishes (Downing and Merkens, 1955).

The percent of un-ionized ammonia in an aqueous ammonia solution increases as the pH increases according to the following equilibria (Butler, 1964).

$$NH_4^+ + OH^- \longleftrightarrow NH_3 \cdot H_2O \longleftrightarrow NH_3 + H_2O$$

The equilibrium between NH_3 and NH_4^+ also shifts toward NH_3 formation as the temperature increases. The combined influence of pH and temperature (°C) on the fraction of total ammonia in the un-ionized form (NH_3) has been described and tabulated by Emerson, et al, (1975) according to the following equation (constants rounded).

$$\text{Fraction of Total Ammonia in Un-ionized Form } (NH_3) = \frac{1}{1 + 10^{(pK\alpha - pH)}} \qquad (1)$$

$$\text{where } pKa = 0.09 + \frac{2730}{273 + T} \qquad (2)$$

and T = °C

The presence of dissolved solids will lower the concentration of un-ionized ammonia slightly. In Great Lakes waters, the magnitude of this effect will usually be less than that of changing the temperature by 1°C under otherwise constant conditions (Whitfield, 1978). The influence of dissolved solids will therefore be ignored in the development of this objective.

Throughout the remainder of this objective un-ionized ammonia (molecular NH_3) concentrations will be referred to as "ammonia". Other forms of ammonia will be indicated where present in the text. Where necessary, un-ionized ammonia concentrations were calculated from the above equation (1).

Occurrence of Ammonia in the Great Lakes

Ammonia occurs naturally in the Great Lakes and represents an integral part of the aquatic ecosystem. Table 2.2.1 identifies typical total ammonia nitrogen levels in mid-lake areas and at selected tributary and channel mouths. The higher ammonia concentrations indicate the presence of active discharges into the littoral zones of the Great Lakes, thereby potentially limiting the use of those areas by aquatic biota.

LETHALITY TO BIOTA

The following sections outline the sensitivity to ammonia of organisms which are representative of major trophic levels in the aquatic environment.

Plants

Plants are not highly sensitive to ammonia nor is nitrogen from this and other sources a limiting nutrient in aquatic systems under aerobic conditions in the Great Lakes.

Ammonium hydroxide addition to a small lake (1.78 ha.) reduced total organism counts of phytoplankton by 84% within 24 hours when ammonia levels peaked at 23 mg/L (Champ, et al., 1973). Phytoplankton were further reduced by 95% after two weeks. Apparent recovery was not observed until four months later (although recovery may have begun earlier) when surface ammonia levels were 0.03 mg/L. The macrophyte American lotus (Nelumbo sp.) began recovery when the mean lake levels dropped to 0.11 mg/L ammonia. Un-ionized ammonia concentrations were calculated from mean lake temperatures and pH reported in the study.

TABLE 2.2.1: Total ammonia nitrogen concentrations (mg/L) in the Great Lakes and at the mouths of tributaries

LAKES	Superior	Huron	Erie	Ontario
Annual Mean(1)	0.007	0.008	0.018	0.018
Tributary Mouth Concentrations(2)	0.05 Agawa R.	0.06-0.11 Mississaga R.	0.05 Sydenham R.	0.14 Hamilton Har.
	0.04 Montreal R.	0.10 Blind R.	0.11 Thames R.	0.14 Toronto Har.
	0.04 Dead Horse R.	0.04-0.26 Serpent R.	0.19 Canard R.	0.08 Oakville Ck.
	0.03-0.11 St. Marys R.	0.02 Saugeen R.	4.28 Turkey R.	0.04 Twelve Mile Ck.
		0.05 Sauble R.	0.08 Nanticoke Ck.	0.06 Twenty Mile Ck.
		0.02 Lucknow R.	0.25 Sandusky Ck.	0.15 Chippawa Canal
		0.08 Ausable R.	0.13 Grand R.	2.78 Welland R.
				0.24 Redhill Ck.
				0.66 Don R.
				0.10 Cataraqui R.

(1) Average annual means (N=10 to 30) 1969-1980 (L. Erie 1979-1980) from Enviroment Canada cruises (data in CCIW STARFILE)
(2) Mean values (N=6 to 12) (OME, 1981).

In another study, algal photosynthesis was inhibited under laboratory conditions in Scenedesmus obliquus, Chlorella pyrenoidosa, Anacystis nidulans and Plectonema boryanum when ammonia levels in water exceeded 2.5 mg/L (Abeliovich and Azov, 1967). These levels of un-ionized ammonia are unusually high and would occur only during major spill situations.

Invertebrates

Champ, et al. (1973) found that cladocerans and copepods did not show signs of recovery until mean lake ammonia levels dropped to 0.02 mg/L, which was slightly less than the level at which phytoplankton demonstrated strong recovery. Rotifers appeared more tolerant than cladocerans and copepods when ammonia levels were 23 mg/L but did not show signs of recovery until ammonia dropped to 0.03 mg/L. Champ, et al. (1973) also reported recovery of crayfish (Cambarus sp.) and freshwater shrimp (Palaemonetes kadiakensis) at 0.02 mg NH_3/L in their whole lake ammonium addition study.

The whole lake study (Champ et al., 1973) makes comparison of invertebrate sensitivities difficult due to the uncontrolled nature of the ammonium hydroxide addition, the lengthy periods between observation times and the un-ionized ammonia concentrations being estimated from mean monthly temperatures and pH, making organism response estimates imprecise.

Laboratory investigations with the rotifer Philodina acuticornis indicated a failure to respond to light when ammonia reached 2.9-9.1 mg/L (Buikema, et al., 1974). Qureshi, et al. (1982) and Parkhurst, et al. (1981) indicated that the 48-hour LC_{50} for Daphnia magna was 3.3 mg NH_3/L (calculated from total ammonia data assuming pH 7.9 and 15°C) and 2.08 mg NH_3/L; respectively. The threshold immobility concentration for Daphnia pulicaria was reported as 1.16 mg NH_3/L (DeGraeve, et al., 1980).

Exposure of fingernail clams (Musculium transversum) to about 0.08 mg NH_3/L, produced 50% mortality after six weeks which coincided with a 50% reduction in ciliary beating of gills in small clams (Sparks, 1981). The same reduction in ciliary beating in larger fingernail clams occurred at about 0.04 mg NH_3/L. Reduced ciliary activity was indicative of potential impairment of food collection, water circulation and waste excretion. Fingernail clams have been recognized as an important food source for diving ducks and bottom-feeding fish in the Illinois River, where the disappearance of clams has been associated in part with elevated ammonia levels.

With the exception of clams, macroinvertebrates and zooplankton do not appear to be adversely affected by ammonia until concentrations exceed about 0.5 mg/L. Fingernail clams appear to be about an order of magnitude more sensitive to ammonia than other invertebrates.

Fish

Lethal levels of ammonia collected from generally available literature for catfish (Ictalurus punctatus), fathead minnow (Pimephales promelas), bluegill sunfish (Lepomis macrochirus), largemouth bass (Micropterus salmoides), striped bass (Morone saxatilis), perch (Perca flavescens),

stickleback (Gasterosteus aculeatus), atlantic salmon (Salmo salar), coho salmon (Oncorhynchus kisutch), cutthroat trout (Salmo clarki), rainbow trout (Salmo gairdneri) and chinook salmon (Oncorhynchus tshawytscha) are presented in Table 2.2.2. Species are listed in order of mean tolerance as are the data within each species.

The majority of reported lethal responses are 96-hour LC_{50}'s and for the larger catfish, rainbow trout and cutthroat trout databases, the 96-hour LC_{50} values overlap those for longer and shorter exposure periods. Comparison of species response ranges indicates that catfish LC_{50}'s bracket those of sticklebacks and striped bass and overlap with largemouth bass and bluegill data. Rainbow trout LC_{50}'s also overlap catfish data but are generally lower. The range of rainbow trout data does, however, overlap largemouth bass, bluegill, fathead minnow, perch, cutthroat trout, chinook, coho and atlantic salmon LC_{50}'s.

While LC_{50}'s are reliable measures of acute lethality, they by definition, provide protection for only half of an exposed population over a short term. Ninety-day exposures of rainbow trout to ammonia established a factor of 0.4 to 0.2 (average 0.3) between threshold mortality (LC_{05}) and a measured 48-hour LC_{50} (Ministry of Technology, 1967). Calculation of a LC_{01} of 0.37 mg NH_3/L for rainbow trout (Broderius and Smith, 1979) represents 0.5 of the 96-hour LC_{50}. Cutthroat trout fry, whose reported sensitivity to ammonia is bracketed by rainbow trout, exhibited a 36-day lethal threshold that ranged from 0.33 to 0.68 of the 96-hour LC_{50} (Thurston, et al., 1978).

The most relevant study to establish a lethal threshold/96-hour LC_{50} ratio is that of Broderius and Smith (1979) with a 0.5 value. However, longer term studies generated lower ratios of 0.2 and 0.33 while Thurston and Russo (1983) have identified larger (1-2.5 kg) rainbow trout as being twice as sensitive to ammonia as are smaller (1-10 g) trout. The greater sensitivity of larger and longer exposed fish supports the use of a lower lethal threshold/LC_{50} ratio of 0.3 to provide additional protection in estimating non-lethal concentrations.

Water Quality Effects on Lethality

Dissolved Oxygen

Merkens and Downing (1957) demonstrated that the acute lethality of ammonia to rainbow trout would increase about four-fold when the dissolved oxygen in water was reduced from 8.8 to 4 mg/L. Lloyd (1961) reported only a 2.5-fold increase in sensitivity of rainbow trout exposed to ammonia when oxygen was reduced to a 40% saturation. Atlantic salmon smolts also exhibited greater sensitivity to ammonia (about two times) when oxygen levels were reduced to 3.5 mg/L (Alabaster, et al., 1979). Thurston, et al. (1981 b) reported a positive correlation between dissolved oxygen and ammonia LC_{50}'s with rainbow trout but did not provide a quantitative relationship for general application. Other water

TABLE 2.2.2: FISH LETHALITY DATA

SPECIES	LIFE STAGE OR SIZE	T E S T	TEST CONDITIONS					CONCENTRATION	REFERENCE
			pH	Temp (°C)	O_2(mg/L)	Alk (mg/L)	Hard (mg/L)	(mg NH_3/L)	
Stickleback	20-93mm	96-hr LC_{50}	7	15	5	N/A	175	3.4	Hazel, et al., 1971
(Gasterosteus aculeatus)	20-93mm	96-hr LC_{50}	7.2	23	5	N/A	175	2.3	Hazel, et al., 1971
Striped Bass	32-60mm	96-hr LC_{50}	7	15	5	N/A	28	2.6	Hazel, et al., 1971
(Morone saxatilis)	32-60mm	96-hr LC_{50}	7.2	23	5	N/A	28	2.2	Hazel, et al., 1971
Channel Cat	50-76mm	96-hr LC_{50}	8.7	30	7.6	220	102	3.8	Colt and Tchobanoglous, 1976
(Ictalurus punctatus)	4.5-8.3g	96-hr LC_{50}	8	28	6.7	301	327	3.6	Roseboom and Richey, 1977
	50-76mm	96-hr LC_{50}	8.7	26	7.6	220	102	2.9	Colt and Tchobanoglous, 1976
	33 g	24-hr LC_{50}	8.7	25	8.3	450		2.87	Robinette, 1976
	50-76mm	96-hr LC_{50}	8.7	22	7.6	220	102	2.4	Colt and Tchobanoglous, 1976
	7-13cm	24-hr LC_{50}	8	23	7	47	40	2.21	Tomasso, et al., 1980
	7-13cm	24-hr LC_{50}	9	23	7	47	40	1.81	Tomasso, et al., 1980
	12.7-7.4g	96-hr LC_{50}	8	22	7.5	301	327	1.8	Roseboom and Richey, 1977
	7-13cm	24-hr LC_{50}	7	23	7	47	40	1.69	Tomasso, et al., 1980
	1.1 g	96-hr LC_{50}	8.4	28	7.6	256	120	1.6	Colt and Tchobanoglous, 1978
	12.7cm	7-day LC_{50}	7.7	22	6	N/A	N/A	1.02	Knepp and Arkin, 1973
Largemouth Bass	.24g	96-hr LC_{50}	8	30	7	283	294	1.5	Roseboom and Richey, 1977
(Micropterus salmoides)	3.59g	96-hr LC_{50}	7.9	22	8	283	294	0.87	Roseboom and Richey, 1977
Bluegill	.27g	96-hr LC_{50}	8.2	28	7	310	330	1.6	Roseboom and Richey, 1977
(Lepomis macrochirus)	.5 g	96-hr LC_{50}	8	22	8	297	323	0.59	Roseboom and Richey, 1977
Fathead Minnow	1.9 g	96-hr LC_{50}	8.5	13.5	9.6	175	182	1.68	Thurston, et al., 1981c
(Pimiphales promelas)	1.9 g	96-hr LC_{50}	9	13.5	9.6	185	182	1.47	Thurston, et al., 1981c
	1.9 g	96-hr LC_{50}	7.8	12	9.6	163	182	1.08	Thurston, et al., 1981c
	1.9 g	96-hr LC_{50}	7.8	11.8	9.6	168	182	0.79	Thurston, et al., 1981c
	1.9 g	96-hr LC_{50}	7	13.8	9.6	133	182	0.45	Thurston, et al., 1981c
	1.9 g	96-hr LC_{50}	6.5	13	9.6	90	182	0.24	Thurston, et al., 1981c
Rainbow Trout	8±7 cm	8-hr LC_{50}	8	20	8.8	240	305	1.96	Merkins and Downing, 1957
(Salmo gairdneri)	8±7 cm	168-hr LC_{50}	8	20	8.8	240	305	1.69	Merkins and Downing, 1957
	0.9 g	96-hr LC_{50}	8	15	10	91	143	1.44	Craig, 1983
	1.2 g	96-hr LC_{50}	8	15	10	80	140	1.18	Craig, 1983
	1.0 g	96-hr LC_{50}	8	15	10	91	143	1.07	Craig, 1983
	5.6cm	8-hr LC_{50}	8.2	20	N/A	250	305	1.02	Lloyd and Herbert, 1960
	2.9 g	96-hr LC_{50}	7.6	15	10	40	82	1.02	Craig, 1983
	1.2 g	96-hr LC_{50}	8	15	10	80	140	1	Craig, 1983
	2.3 g	96-hr LC_{50}	7.8	12	8.6	172	205	0.81	Thurston, et al., 1981b
	4-5 mo.	96-hr LC_{50}	8.3	14	7.8	188	200	0.8	Thurston, and Russo, 1983
	0.5 g	96-hr LC_{50}	7.6	15	10	40	82	0.76	Craig, 1983
	5.7 g	96-hr LC_{50}	7.8	13	7.7	172	205	0.76	Thurston, et al., 1981b

continued

TABLE 2.2.2: FISH LETHALITY DATA (continued)

SPECIES	LIFE STAGE OR SIZE	TEST	TEST CONDITIONS pH	Temp (°C)	O_2(mg/L)	Alk (mg/L)	Hard (mg/L)	CONCENTRATION (mg NH_3/L)	REFERENCE
Rainbow trout (Salmo gairdneri)	5.6 cm	8-hr LC_{50}	7.8	20	N/A	250	305	0.75	Lloyd and Herbert, 1960
	4-5 mo	96-hr LC_{50}	7.9	13	7.8	175	200	0.71	Thurston and Russo, 1983
	1.7 g	96-hr LC_{50}	7.8	12	8.6	172	205	0.7	Thurston, et al., 1981b
	5.7 g	96-hr LC_{50}	7.8	13	7.4	172	205	0.7	Thurston, et al., 1981b
	4-5 mo	96-hr LC_{50}	8.8	14	7.8	193	200	0.68	Thurston and Russo, 1983
	4 g	96-hr LC_{50}	7.8	13	7.6	172	205	0.68	Thurston, et al., 1981b
	0.5 g	96-hr LC_{50}	7.8	15	10	93	137	0.67	Craig, 1983
	4-5 mo	96-hr LC_{50}	9	15	7.8	196	200	0.65	Thurston and Russo, 1983
	4-5 mo	96-hr LC_{50}	7.8	13	7.9	177	200	0.64	Thurston and Russo, 1983
	3.2 g	96-hr LC_{50}	7.8	13	5.5	172	205	0.64	Thurston, et al., 1981b
	8.5-14.5 cm	48-hr LC_{50}	7.8	11	10	240	320	0.62	Brown, et al., 1969
	yearling	48-hr LC_{50}	6.9	17.7	9	41.5	44	0.62	Herbert and Shurben, 1964
	yearling	48-hr LC_{50}	7.8	17.7	9	240	320	0.62	Herbert and Shurben, 1964
	0.6 g	96-hr LC_{50}	7.8	15	10	93	137	0.62	Craig, 1983
	4-5 mo	96-hr LC_{50}	7.8	13	7.8	176	200	0.62	Thurston and Russo, 1983
	5.6 cm	8-hr LC_{50}	7	20	N/A	250	305	0.6	Lloyd and Herbert, 1960
	12.3 cm	24-hr LC_{50}	7.8	12	7	240	320	0.6	Herbert and Shurben, 1963
	5 g	96-hr LC_{50}	7.8	13	6.6	172	205	0.6	Thurston, et al., 1981b
	8±7 cm	8-hr LC_{50}	8	20	4	240	305	0.58	Merkins and Downing, 1957
	5.7 g	96-hr LC_{50}	7.8	13	6.6	172	205	0.56	Thurston, et al., 1981b
	11.3 cm	24-hr LC_{50}	7.8	12	7	240	320	0.55	Herbert and Shurben, 1963
	2.3 g	96-hr LC_{50}	7.8	12	4.4	172	205	0.54	Thurston, et al., 1981b
	12.5 cm	48-hr LC_{50}	7.8	17	10	250	320	0.52	Herbert and Van Dyke, 1964
	35-95 g	96-hr LC_{50}	7.9	15	10	N/A	N/A	0.52	Smart, 1976
	1.7 g	96-hr LC_{50}	7.8	13	4.4	172	205	0.52	Thurston, et al., 1981b
	5.6 cm	8-hr LC_{50}	7.4	20	N/A	250	305	0.51	Lloyd and Herbert, 1960
	11.3 cm	24-hr LC_{50}	7.8	12	7	240	320	0.51	Herbert and Shurben, 1963
	4.5 g	96-hr LC_{50}	7.2	15	10	40	82	0.51	Craig, 1983
	40 g	48-hr LC_{50}	8.04	11	10	230	290	0.5	Ball, 1967
	4.6 g	96-hr LC_{50}	7.8	13	5.7	172	205	0.5	Thurston, et al., 1981b
	21 g	96-hr LC_{50}	7.9	9.7	8.7	170	189	0.5	Thurston, et al., 1981a
	9.4 g	96-hr LC_{50}	7.9	13	3.6	172	205	0.48	Thurston, et al., 1981b
	8±7 cm	168-hr LC_{50}	8	20	4	240	305	0.47	Merkins and Downing, 1957
	N/A	48-hr LC_{50}	N/A	N/A	N/A	N/A	290	0.45	Min. of Tech., 1967
	4-5 mo	96-hr LC_{50}	7.3	14	7.8	149	200	0.45	Thurston and Russo, 1983
	10.1 g	96-hr LC_{50}	7.8	13	3.2	172	205	0.4	Thurston, et al., 1981b
	17-23 cm	24-hr LC_{50}	8.1	10.5	9	240	320	0.39	Lloyd and Orr, 1969
	2.9 g	96-hr LC_{50}	7.1	15	10	4	15	0.39	Craig, 1983
	8.8 g	96-hr LC_{50}	7.9	13	2.7	172	205	0.33	Thurston, et al., 1981b
	8.2 g	96-hr LC_{50}	7.9	13	2.6	172	205	0.32	Thurston, et al., 1981b

continued

TABLE 2.2.2: FISH LETHALITY DATA (continued)

SPECIES	LIFE STAGE OR SIZE	TEST	TEST CONDITIONS					CONCENTRATION	REFERENCE
			pH	Temp (°C)	O_2(mg/L)	Alk (mg/L)	Hard (mg/L)	(mg NH_3/L)	
Rainbow trout (Salmo gairdneri)	0.6 g	96-hr LC_{50}	6.3	15	10	3.8	8	0.3	Craig, 1983
	247 g	96-hr LC_{50}	7.7	8.1	8.7	170	189	0.3	Thurston, et al., 1981a
	sac-fry	7-day LC_{50}	7.5	11	8	105	115	0.27	Burkhalter and Kaya, 1977
	sac-fry	21-day LC_{50}	7.5	11	8	105	115	0.25	Burkhalter and Kaya, 1977
	2.9 g	96-hr LC_{50}	7.1	15	10	4	15	0.19	Craig, 1983
	4-5 mo	96-hr LC_{50}	6.8	14	7.8	107	200	0.19	Thurston and Russo, 1983
	4-5 mo	96-hr LC_{50}	6.5	14	7.8	75	200	0.16	Thurston and Russo, 1983
	2596 g	96-hr LC_{50}	7.6	7.9	8.7	170	189	0.16	Thurston, et al., 1981a
	1.7 g	96-hr LC_{50}	6.6	15	10	5.1	5.6	0.13	Craig, 1983
	1.7 g	96-hr LC_{50}	6.6	15	10	5.1	5.6	0.1	Craig, 1983
	adult	24-hr LC_{50}	8.3	10	10	0.05 M TRIS BUFFER USED		0.097	Rice and Stokes, 1975
	85 day fry	24-hr LC_{50}	8.3	10	13	0.05 M TRIS BUFFER USED		0.068	Rice and Stokes, 1975
Chinook Salmon (Oncorhynchus tshawytscha)	juvenile	96-hr LC_{50}	8.5	15	N/A	100	50/115	0.88	Robinson-Wilson and Seim, 1975
	juvenile	96-hr LC_{50}	8	15	N/A	100	50/115	0.71	Robinson-Wilson and Seim, 1975
	juvenile	96-hr LC_{50}	8	15	N/A	100	50/115	0.7	Robinson-Wilson and Seim, 1975
	juvenile	96-hr LC_{50}	7.5	15	N/A	100	50/115	0.55	Robinson-Wilson and Seim, 1975
	juvenile	96-hr LC_{50}	7.5	15	N/A	100	50/115	0.53	Robinson-Wilson and Seim, 1975
	juvenile	96-hr LC_{50}	7	15	N/A	100	50/115	0.28	Robinson-Wilson and Seim, 1975
	juvenile	96-hr LC_{50}	7	15	N/A	100	50/115	0.27	Robinson-Wilson and Seim, 1975
Coho Salmon (Oncorhynchus kisutch)	6 g	96-hr LC_{50}	7-9	17	8	38/136	37	0.55	Buckley, 1978
Cutthroat Trout (Salmo clarki)	1 g	96-hr LC_{50}	7.8	13.1	8.6	176	199	0.8	Thurston, et al., 1978
	1 g	96-hr LC_{50}	7.8	12.8	8.4	176	199	0.66	Thurston, et al., 1978
	3.3 g	96-hr LC_{50}	7.8	12.4	8.2	176	199	0.62	Thurston, et al., 1978
	1 g	36-day LC_{50}	7.8	13.1	8.6	176	199	0.56	Thurston, et al., 1978
	1 g	36-day LC_{50}	7.8	12.8	8.4	176	199	0.56	Thurston, et al., 1978
	3.4 g	96-hr LC_{50}	7.8	12.2	8.3	176	199	0.52	Thurston, et al., 1978
	3.3 g	29-day LC_{50}	7.8	12.4	8.2	176	199	0.37	Thurston, et al., 1978
	3.4 g	29-day LC_{50}	7.8	12.2	8.3	176	199	0.34	Thurston, et al., 1978
	3.6 g	96-hr LC_{50}	7.7	10	8.7	170	189	0.33	Thurston, et al., 1981a
	4.1 g	96-hr LC_{50}	7.7	10	8.7	170	189	0.3	Thurston, et al., 1981a
Perch (Perca flavescens)	14 g	24-hr LC_{50}	7.9	10	11	230	290	0.35	Ball, 1967
Atlantic Salmon (Salmo salar)	2+ yrs	24-hr LC_{50}	7.7	12	9.6	N/A	264	0.145	Alabaster, et al., 1979
	2+ yrs	24-hr LC_{50}	7.7	12	3.5	N/A	264	0.086	Alabaster, et al., 1979

quality factors such as carbon dioxide effects (Alabaster and Herbert, 1955; Lloyd and Herbert, 1960; Lloyd, 1961) influencing ammonia toxicity, continue to prohibit adequate quantification of the O_2-NH_3 relationship. The above studies indicate, however, that low dissolved oxygen concentrations increase the toxicity of ammonia and underline the importance of maintaining dissolved oxygen at the current minimum objective of 6.0 mg/L. This value itself may not be sufficiently protective at temperatures of 5°C and less when conditions represent less than 50% saturation.

Temperature

A few researchers have described an increase in ammonia toxicity to fish at low water temperatures. Largemouth bass, bluegills and channel catfish LC_{50}'s have been reduced by about one half when exposed to ammonia at 22°C, compared to 30°C (Colt and Tchobanoglous, 1976; Roseboom and Richey, 1977). Nimmo (personal communication) has also described an increase in sensitivity to ammonia under low (1-5°C) temperature conditions.

While temperature apparently affects the toxicity of ammonia to fish, the relationship has not been developed clearly for application among all species and therefore cannot be included in the calculation of an objective. In consideraton of the seasonal wide range of temperature fluctuations in the Great Lakes, this influence requires quantificaton.

Hydrogen Ion (pH)

Test pHs in most studies of ammonia toxicity have mainly been employed in the calculation of the more toxic un-ionized component (NH_3). Few studies have investigated the effect of pH on NH_3 toxicity *per se*. Thurston, *et al*. (1981 c) described a positive relationship between pH and 96-hour NH_3 LC_{50}'s for fathead minnows and rainbow trout. Craig (1983) described a similar increase of NH_3 toxicity under low pH and low alkalinity conditions during an interlaboratory calibration exercise using rainbow trout. The effect of increased ammonia toxicity under low alkalinity conditions was supported by multiple regression analysis only and may have been a reflection of the pH effect.

Erickson (1982), drawing on published data, developed NH_3 toxicity-pH relationships for *Daphnia*, rainbow trout, fathead minnow and coho salmon as follows:

$$LC_{50} = \frac{X}{1 + 10^{1.03(7.32-pH)}} \qquad (3)$$

This formula describes a toxicity-pH relationship which changes rapidly in the low pH range but becomes nonlinear and levels off at high pH. The X value is the highest LC_{50} at elevated pH where the slope of the relationship approaches zero. Since species differ in their sensitivity

to ammonia, the value for X is species dependent. The X value for the most sensitive species, rainbow trout, was 0.66 mg NH_3/L (Erickson, 1982). Using this relationship, the LC_{50} for rainbow trout exposed to ammonia under a range of pH conditions can be estimated.

SUBLETHAL EFFECTS ON FISH

The following outline of sublethal effects is limited to those reported for fish (Table 2.2.3) since the few sublethal responses documented for other organisms were included in the previous section.

Egg Viability and Hatching

Rainbow trout egg viability was unaffected after a 25-day exposure to 0.45 mg NH_3/L; however, alevins developed blue sac disease when exposed to 0.23 mg NH_3/L (Burkhalter and Kaya, 1977). Rainbow trout eggs were also unaffected by a 24-hour exposure of 3.58 mg NH_3/L while the 24-hour LC_{50} for fry and adults under the same test conditions was 0.068 and 0.097 mg/L (Rice and Stokes, 1975). The 62-day LC_{50} for sockeye salmon eggs was reported at 0.13 mg NH_3/L, but this included a TRIS buffer effect that was estimated at 9% (Rankin, 1979). In fact, the buffer-control egg mortality was double that of the unbuffered control treatment. Rice and Stokes (1975) also used TRIS buffer to increase the exposure pH but did not use a buffer-control in their experimental design. These studies indicate that fish eggs are probably less sensitive to ammonia than are juvenile and adult fish and therefore would be protected by the same nonlethal objective requirement.

Growth

Rainbow trout growth impairment has been reported at 0.06 mg NH_3/L for fry after 42 days' exposure (Burkhalter and Kaya, 1977), and at 0.033 mg NH_3/L after 6 months' exposure (Smith, 1972). A level of 0.025 mg/L had no effect on rainbow trout growth (Smith 1972). Thurston, et al. (1984) observed that rainbow trout growth was not impaired after four years' exposure to 0.07 mg NH_3/L.

Growth impairment in chinook salmon has been reported at 0.05 mg NH_3/L with no effect at 0.03 mg NH_3/L (Robinson-Wilson and Seim, 1975). Chinook growth was unaffected at 0.017 mg NH_3/L when dissolved oxygen was 7 mg/L or higher but was impaired when dissolved oygyen was less than 5 mg/L (Larmoyeux and Piper, 1973).

The significance of available oxygen becomes critical when reviewing reports on growth effects. Burrows (1964) claimed that chinook salmon growth was reduced when ammonia exceeded 0.006 mg/L. However, the percent ionized ammonia was not calculated using accurate ionization constants (Trussel, 1972), growth data were not presented and dissolved oxygen levels were not reported. The over-riding influence of oxygen on growth in higher ammonia concentrations (Larmoyeux and Piper, 1973) than those reported by Burrows (1964), suggests that the response attributed to ammonia in the latter case may have been confounded by other factors such as reduced oxygen.

TABLE 2.2.3: FISH SUBLETHAL DATA

SPECIES	LIFE STAGE OR SIZE	RESPONSE	TEST CONDITIONS					CONCENTRATION	REFERENCE
			pH	Temp (°C)	O_2(mg/L)	Alk (mg/L)	Hard (mg/L)	(mg NH_3/L)	
Channel Cat	20-160 g	no growth	8.4	28	7.6	256	120	1.2	Colt and Tchobanoglous, 1978
(Ictalurus punctatus)	33 g	reduced growth	7.8	25	5.1	450	N/A	0.15*	Robinette, 1976
	33 g	no-effect growth	7.8	25	5.1	450	N/A	0.07*	Robinette, 1976
	20-160 g	reduced growth	8.4	28	7.6	256	120	0.06	Colt and Tchobanoglous, 1978
Sockeye Salmon	eggs	62-day LC_{50}	8.4	10	13	0.05 M TRIS BUFFER USED		0.13	Rankin, 1979
(Oncorhynchus nerka)									
Bluegill sunfish	5-7.5 cm	EC_{60} avoidance	7/7.4	20/22	7.8	20/40	28/40	0.05	Lubinski, et al., 1980
(Leponis macrochirus)	5-7.5 cm	no avoidance	7/7.4	14	7.8	20/40	28/40	0.05	Lubinski, et al., 1980
	5-7.5 cm	no avoidance	7/7.4	20/22	7.8	20/40	28/40	0.005	Lubinski, et al., 1980
Chinook Salmon	juvenile	impaired growth	7.5	15	N/A	100	50/115	0.05*	Robinson-Wilson and Seim, 1975
(Oncorhynchus	juvenile	no-effect growth	7.5	15	N/A	100	50/115	0.03*	Robinson-Wilson and Seim, 1975
tshawytscha)	3-15 cm	reduced growth	7.7	N/A	5	185	185	0.017*	Larmoyeux and Piper, 1973
	3-15 cm	no-effect growth	7.7	N/A	7	185	185	0.017	Larmoyeux and Piper, 1973
	fingerlings	gill pathology	7.8	6/14	N/A	N/A	N/A	0.0026*	Burrows, 1964
Rainbow trout	egg	24-hr no effect	8.3	10	11	0.05 M TRIS BUFFER USED		3.58	Rice and Stokes, 1975
(Salmo gairdneri)	fertilize	24-hr no effect	8.3	10	11	0.05 M TRIS BUFFER USED		1.79	Rice and Stokes, 1975
	egg survival	no effect	7.5	10/12	13	105	106/123	0.45	Burkhalter and Kaya, 1977
	35-85 g	gill pathology	7.9	15	10	N/A	N/A	0.33	Smart, 1976
	yolk sac fry	blue sac	7.5	10/12	13	105	106/123	0.23	Burkhalter and Kaya, 1977
	42 day fry	gill pathology	7.5	10/12	13	105	106/123	0.23	Burkhalter and Kaya, 1977
	adult	no effect growth	7.7	9.3	7.5	173	197	0.07	Thurston, et al., 1984
	fry	impaired growth	7.5	11	8	105	115	0.06	Burkhalter and Kaya, 1977
	17-23 cm	diuretic	8.1	10.5	9	240	320	0.056	Lloyd and Orr, 1969
	adult	kidney path.	7.7	9.3	7.5	173	197	0.04	Thurston, et al., 1984
	1.6-120 g	impaired growth	7.75	10	5.9	N/A	200	0.033	Smith, 1972
	1.2-134 g	no-effect growth	7.75	10	6.4	N/A	200	0.025	Smith, 1972
Pink Salmon	61-day fry	reduced growth	6.4	3.7-4.8	sat?	N/A	N/A	0.0024**	Rice and Bailey, 1980
(Oncorhynchus	61-day fry	no-effect growth	6.4	3.7-4.8	sat?	N/A	N/A	0.0012**	Rice and Bailey, 1980
gorbuscha)									

* - Dissolved oxygen less than the IJC Objective of 6 mg/L or unknown therefore data not used to develop objective.
** - Undefined water quality, low temperatures and data unsupported by other studies resulted in data not being used to develop objective.

Rice and Bailey (1980) also reported a low ammonia concentration growth impairment value of 0.0024 mg/L and a no-effect concentration of 0.0012 mg/L for pink salmon. Under the test conditions of pH 6.4 and 4.5°C using $(NH_4)_2SO_4$, only 0.0005 of the total ammonia would be ionized. These values under more realistic Great Lakes conditions (pH= 8, T=10°C) would result in levels of approximately 0.09 and 0.04 mg/L ammonia, respectively.

There is some disagreement in the above report with respect to exposure concentrations. The 0.0024 mg/L value is exceptionally low (about an order of magnitude lower than other reported values) and is unsupported by other studies. Also, the dilution water was typical of coloured Alaskan muskeg waters high in organic content (Bailey personal communication) which further differentiates the result from those relevant to Great Lakes waters.

Channel catfish fingerlings cultured by Robinette (1976) showed significant growth retardation at 0.15 mg NH_3/L, although the highest no-effect concentration was 0.07 mg NH_3/L. Colt and Tchobanoglous (1978) reported reduced growth of catfish at 0.06 mg NH_3/L.

Review of the above acceptable salmonid growth data indicates that the lowest effect concentrations (0.06, 0.033, 0.05 mg/L) are very close to and indeed are overlapped by the highest no-effect concentrations (0.025, 0.07, 0.03, 0.017 mg/L). Examination of these data suggest that ammonia concentrations of 0.03 mg/L or less would be protective of salmonid growth.

Other Sublethal Effects

Gill lamellar swelling (hyperplasia) and abnormally high surface cell growth (epithelial proliferation) have been observed in rainbow trout exposed to 0.33-0.23 mg NH_3/L (Smart, 1976; Larmoyeux and Piper, 1973). Gills developed proliferation of epithelium, hyperplasia of lamellae (resulting in fusion) and blood pooling (cystic haematomas) and tissue death (necrosis) in sequence as NH_3 levels increased. Pathological damage to gills predisposes fish to epizootic bacterial gill disease and impairs oxygen utilization (Smart, 1978). Gill damage due to elevated ammonia concentrations has been qualitatively related to growth (Burrows, 1964).

The diuretic effect on fish exposed to 0.056 mg/L ammonia suggests increased gill permeability (Lloyd and Orr, 1969). Lethal levels of ammonia also increased ammonia in rainbow trout blood but did not appear to affect the ability of oxygen to combine with haemoglobin (Fromm and Gillette, 1968). Generalized nephrosis, degeneration of renal tubule epithelia, hyaline droplet degeneration and some occluded tubule lumens were observed in rainbow trout exposed for four years to 0.04 mg NH_3/L (Thurston, et al., 1984).

Acute lethal exposures of rainbow trout to 0.5 and 0.65 mg NH_3/L produced increases in oxygen consumption (threefold), ventilation volume, respiratory frequency and amplitude, heart rate and dorsal aortic blood pressure with a simultaneous decrease in dorsal aortic PO_2 (Smart, 1978). It was suggested that, since gill oxygen transfer ability did not appear to be affected, the blood's oxygen transport capability may have been impaired, or alternatively that cellular metabolism was altered so as to increase oxygen requirements.

Lubinski, et al. (1980) monitored the swimming patterns of individual bluegills after exposure to sublethal gradients of ammonia. At levels of 0.05 mg NH_3/L, the fish exhibited a brief drop in activity followed by variable preference-avoidance responses which appeared to be temperature dependent. At 22°C sixty percent of the fish avoided the 0.05 mg/L ammonia while at 14°C there was no response.

Sublethal levels of ammonia can produce pathological changes in the gill, an increase in the susceptibility to bacterial infection, respiratory and cardiac hyperactivity, measurable changes in critical behavioral responses and lower energy output. These pathological effects further support the limited data base available for growth and indicate the presence of stress at various levels which have not been well quantified but represent a variety of additional sublethal responses. These responses occur at comparable levels to those observed to cause growth impairment.

TOXIC EFFECTS OF AMMONIA TO HUMANS

Contact with anhydrous liquid ammonia or with aqueous solutions of concentrations over 500 mg/L is intensely irritating to mucous membranes, eyes and skin (NIOSH, 1974). Higher concentrations of aqueous ammonia may produce serious eye damage including increased intraocular pressure, corneal ulceration or corrosive burns or blisters on the skin.

Ammonia gas is also irritating to the eyes and moist skin. High concentrations result in dyspnea and pulmonary edema, as well as intense irritaton of mucous membranes (NIOSH, 1974). Severe acute exposure to ammonia gas may result in airway damage and reduced gas transfer for up to three years.

Dilute ammonia solutions in contact with chlorinated waters produce chloramines. These compounds are soluble in water and decompose to ammonia and hypochlorous acid. The fumes of chloramines, in significant concentrations, produce tearing and irritation of respiratory and intestinal mucosa. Long term follow-up, however, indicates no permanent damage.

Acute intoxication by ammonium ion, however, resembles the clinical picture of terminal liver failure. Incidents of intoxication are a result of ingestion of fresh "household ammonia" (1-3% ammonia solution), or the misuse of ammonium chloride through accidental addition of bleach to ammonia cleaning fluids (Done and Ozera, 1966; Faigel, 1964).

In general, inhalation or ingestion of ammonia does not produce signs of systemic intoxication and there do not appear to be significant cumulative effects of chronic ammonia exposure (Weatherby, 1952). Further, humans have a high capacity to remove ammonia through the synthesis and excretion of urea. Ammonia does, however, produce headache, insomnia, nausea and diarrhea in humans after ingestion of large quantities (50-100 g) over 3 to 5 days. Other deleterious effects in man unique to ammonia are not known (Health and Welfare Canada, 1978).

OBJECTIVE DEVELOPMENT

Ammonia is non-persistent and is a nutrient in the aquatic environment. It is non-accumulative and has a relatively short-acting respiratory toxicity to fish.

The existing IJC objective of 0.02 mg/L un-ionized ammonia for the protection of fresh water aquatic life is generally less than the drinking water level of 0.5 mg/L total ammonia. The drinking water objective was intended to provide an ammonia limit for raw water due to the reactivity of ammonia with chlorine, which is commonly added at treatment plants for disinfection purposes. The 0.5 mg/L total ammonia drinking water level does not have toxic effects on humans.

Laboratory studies have demonstrated that fish can tolerate short term exposure to potentially lethal ammonia concentrations and return to sublethal or non-lethal conditions with no apparent detrimental effect (Brown, et al., 1969; Thurston, et al., 1981 a). This evidence provides justification for permitting relatively high levels of ammonia to exist for short periods (< 96 hours) without injury to sensitive target organisms. The most likely areas for these concentrations to occur is where limited use zones overlap zones of passage. Concentrations for threshold lethality can therefore be tolerated in keeping with the guidelines for limited use zones (IJC, 1974).

To protect the most sensitive species from short term (< 96 hours) exposure to ammonia, where limited use zones overlap routes of passage, the estimated lethal threshold concentration of un-ionized ammonia for rainbow trout must not be exceeded. This requirement can be expressed by:

$$\text{mg un-ionized ammonia/L} = 0.3 \times \frac{0.66}{1 + 10^{1.03(7.32-pH)}} \qquad (4)$$

where pH is the pH within the limited use zone.

Table 2.2.4 lists un-ionized ammonia values calculated according to the above equation (4) for various pH and temperature conditions with equivalent values for total ammonia as nitrogen estimated using equations 1 and 2.

The most sensitive response of aquatic biota reported in the literature is that of growth in the salmonid family. The highest no-effect concentration reported for this family is 0.03 mg NH_3/L. This level will also provide adequate protection for raw water supplies of drinking water. Equivalent values of total ammonia as nitrogen for various pH and temperature conditions appear in Table 2.2.5.

TABLE 2.2.4: OBJECTIVE LEVELS OF UNIONIZED AMMONIA IN LIMITED USE ZONES.
(mg-N/L)

Zone pH	6.50	7.00	7.50	8.00	8.50	9.00
Objective	0.03	0.06	0.12	0.17	0.19	0.20
Temperature (°C)	E Q U I V A L E N T		T O T A L		A M M O N I A	
0.00	80.10	64.67	38.88	17.03	6.20	2.15
5.00	53.01	42.76	25.73	11.30	4.15	1.48
10.00	35.56	28.69	17.29	7.63	2.83	1.04
15.00	24.19	19.53	11.79	5.23	1.98	0.76
20.00	16.67	13.47	8.15	3.65	1.41	0.57
25.00	11.64	9.42	5.72	2.59	1.03	0.45
30.00	8.22	6.66	4.07	1.87	0.77	0.37

TABLE 2.2.5: OBJECTIVE EQUIVALENT LEVELS OF TOTAL AMMONIA.
(equivalent to 0.03 mg un-ionized ammonia/L)

pH	6.50	7.00	7.50	8.00	8.50	9.00
Temperature (°C)	E Q U I V A L E N T		T O T A L		A M M O N I A (mg-N/L)	
0.00	96.14	30.42	9.64	3.06	0.99	0.33
5.00	63.55	20.11	6.38	2.03	0.66	0.23
10.00	42.63	13.50	4.29	1.37	0.45	0.16
15.00	29.00	9.19	2.92	0.94	0.31	0.12
20.00	19.99	6.34	2.02	0.66	0.22	0.09
25.00	13.95	4.43	1.42	0.47	0.16	0.07
30.00	9.86	3.13	1.01	0.34	0.12	0.06

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2.3 BENZENEHEXACHLORIDES (BHC)

EXISTING OBJECTIVE (LINDANE)

The concentration of lindane in water should not exceed 0.01 micrograms per litre for the protection of aquatic life. The concentration of lindane in edible portions of fish should not exceed 0.3 micrograms per gram (wet weight basis) for the protection of human consumers of fish.

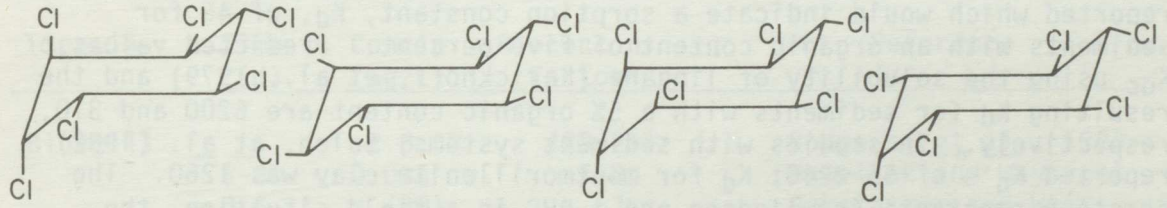
RECOMMENDED OBJECTIVE

The concentration of total hexachlorocyclohexane (BHC) isomers should not exceed 0.02 micrograms per litre for the protection of aquatic life. The concentration of total BHC isomers in edible portions of fish should not exceed 0.3 micrograms per gram for the protection of human consumers of fish.

RATIONALE

Introduction

Lindane is the name given to the gamma-(γ) isomer of benzene hexachloride (BHC) which is also, more properly, named hexachlorocyclohexane (HCCH). There are several other environmentally important isomers, the most important of these being the alpha- (α), beta- (β) and delta- (δ) forms.



α-BHC β-BHC lindane δ-BHC

The commercial mixture of all isomers known as BHC (circa 55-70% α-, 5-14% β-, 10-18% lindane, 6-10% δ-) has been used as a contact insecticide for over thirty years (Colson, 1979). For most of this period and particularly in recent years in North America, the use of lindane has displaced that of technical BHC since most of the insecticidal activity has been attributed to the lindane component (IARC, 1980). Lindane is marketed as a pesticide effective against a broad spectrum of

plant and animal parasites but it has also been shown to have significant impacts on non-target, aquatic species as well. In Canada, it is currently registered as an insecticide and acaricide for domestic or commercial use on vegetable crops, on livestock, in food processing plants and in private and public buildings. In the United States, registered uses include application to fruit, vegetable and field crops as well as general outdoor use and use with livestock.

Physical-Chemical Properties

Lindane is a solid with a melting point of 112 °C. It is generally stable to heat and acids but will undergo dehydrochlorination with strong alkali (Colson, 1979). It is considerably more water-soluble than many other organochlorine compounds of environmental interest. Lindane has reported solubilities of 6.7-10.3 mg/L (Hansen, 1979; Malaiyandi, et al., 1982; Mills and Biggar, 1969; Staples, et al., 1983) which apparently reduces to 0.5 mg/L if ultrafine (0.04 μ) filtration is employed (Roebeck, et al., 1965). The values for the α-isomer are 1.2-4.3 mg/L (Malaiyandi, et al., 1982; Kurihara, et al., 1973), 0.13-2.7 mg/L for the ß-isomer (Mills and Biggar, 1969; Kurihara, et al., 1973) and 8.6-15.7 mg/L for δ-BHC (Kurihara, et al., 1973). The BHC isomers have low (but significant for environmental purposes) vapour pressures which have been variously reported as 0.94 - 16. x 10^{-5} torr for lindane, 0.028 x 10^{-5} torr for ß-BHC and 2.5 x 10^{-5} torr for the α-isomer at ambient temperatures (Seiber, et al., 1983; Callahan, et al., 1979).

The distribution of a chemical on sediment or soil is customarily determined as K_d (concentration on sediment/concentration in water with both in equilibrium) and K_{OC} (an empirical constant related to K_d by the relationship $K_{OC} = K_d$/fraction of organic carbon content). The partitioning of lindane on sediments is modest; a K_{OC} of 912 has been reported which would indicate a sorption constant, K_d, of 45 for sediments with an organic content of five percent. Predicted values of K_{OC} using the solubility of lindane (Karickhoff, et al., 1979) and the resulting K_d for sediments with a 5% organic content are 6200 and 310, respectively. In studies with sediment systems, Saleh, et al. (1982) reported K_d's of 56-2240; K_d for montmorillonite clay was 1260. The sorption constants for lindane and α-BHC in a field situation, the Niagara River, were both observed to be approximately 1000 (Kuntz and Warry, 1983).

Sorption to soils was studied by Baluja, et al. (1975) who found that lindane was rapidly adsorbed at concentrations directly proportional to the organic content of the soil. On soils ranging from bentonite to "peaty muck", Mills and Biggar (1969) found lindane sorption constants of 3-330 while those for the ß-isomer were 4-456. A K_d of 170-180 was observed for sandy as well as for loam soils (Fuhremann and Lichtenstein, 1980). Hamelink and Waybrant (1976), in their quarry system, reported data which indicated sediment sorption constants of 75-240.

The sorption to soil, however, is not irreversible and volatilization occurs, the exact rate being a function of the moisture content of the soil (Spencer and Cliath, 1970) and the soil type (Guenzi and Beard, 1970). Volatilization from aqueous solutions (0.01-1 mg/L) also occurs with half-lives for lindane and α-, β- and δ-BHC being reported at 5-12 days, 4-6 days, 4-9 days and 10-22 days, respectively (Kawahara, 1972). Sidaramappa and Sethunathan (1976) observed a comparable rate with lindane (half-life approximately 3 days) which was also the rate of loss from sandy soil; alluvial soil was 4 or 18 days depending on whether it was flooded or not.

The fugacity-type approach (Mackay and Paterson, 1982) has been applied to the estimation of environmental distribution using the preceding data and data on accumulation and on the kinetics of degradation processes. For equilibrium among the compartments of air, water, soil, sediment, suspended solids and fish, most of the lindane is likely to be found in the sediments (79%), followed by soil (17%) and water (4%) with only trace quantities in the other compartments. Concentrations, however, are expected to be highest in suspended solids and fishes.

Accumulation in Biota

Octanol-water partition coefficients of 5200-7900 have been reported (Elgar, 1983) and this range would predict fish bioconcentration factors of 250-410 (Veith, et al., 1979; Mackay, 1982). A correlation between solubility and the bioconcentration factor for Daphnia magna (Geyer, et al., 1981) predicts a factor of 500-600 using the data above. Partitioning into biota (fishes) has also been investigated in laboratory systems and some values are reported in Table 2.3.1.

Table 2.3.1. Bioconcentration Factors for Lindane in Fishes

Species	Time (d)	Conc'n (µg/L)	Bioconc'n Factor	Reference
Bluegill	6	2.8,32	420,211	Rodgers, et al., 1983
	5-81	0.035 av.	768	Hamelink and Waybrant, 1976
	0.5-1	(30)	67	Gakstatter and Weiss, 1967
Guppy	33	(>2)	560	Metcalf, et al., 1973
Goldfish	0.5-1	(30)	77	Gakstatter and Weiss, 1967
Fathead minnow	304	1.4-23.5	284-674	Macek, et al., 1976
Rainbow trout	(110)	0.018 av.	486	Hamelink and Waybrant, 1976

N.B. - Values in parentheses are estimated from data in the reference.

For the guppy (Lebistes reticulatus) in a system which included exposure via both water and food, Canton, et al. (1975) observed a factor of 540 for α-BHC; the same factor without the food was about 135. Another study on accumulation included sorption of lindane on Chlorella cells in a laboratory system; a BCF of 700 on a wet weight or 4000 on a dry weight basis was observed (Hansen, 1979). Hamelink and Waybrant (1976) recorded data indicating a zooplankton concentration factor of 170-448 over 60 days in their quarry system.

Lower bioconcentration factors have been reported to arise as a consequence of low exposure concentrations in laboratory systems. Values of 60 and 360 were observed with Daphnia magna when water levels of α-BHC were 10-50 and 800 µg/L, respectively (Canton, et al., 1975). The applicability of these findings to natural systems involving fishes, however, is not clear. The observations are also not in accord with the work of Rodgers, et al. (1983) with bluegills although they are with the results of Macek, et al. (1976) using fathead minnows.

Degradation and Persistence

The conversion of lindane to the more stable α- and β- isomers of BHC has been reported for microbial and sediment systems (Benezet and Matsumura, 1973) in which some 20% of the α- isomer is estimated to have been formed after 30 days. Similarly, Newland, et al. (1969) found α- and δ- on anaerobic incubation of lindane with sediments. There is little evidence that similar conversions occur photochemically, in either aqueous solution or in the gas phase in anything other than trace amounts (Malaiyandi, et al., 1982; Steinwandter, 1976; Vollner and Korte, 1974).

Abiotic aspects of the persistence of lindane have recently been reported by Saleh, et al. (1982). Hydrolytic half-lives of 0.5-4.6 weeks were observed in a laboratory system using filtered natural waters and sediments from three sources with aqueous pH's of 7.3-9.3. Photolysis under similar conditions gave half-lives of 1.0-10.7 weeks, figures which were approximately halved when allowance was made for mid-winter light conditions. The rates of both hydrolysis and photolysis were observed to be retarded relative to those in distilled water when natural waters of comparable pH were employed. This suggests an association of the lindane with material (probably organic) dissolved in the natural water.

There are a number of studies reporting on the degradation or loss of lindane in the environment. Most of them pertain to soil systems but some are relevant to aquatic ecosystems. Yoshida and Castro (1970) examined the fate of lindane in a laboratory system employing flooded rice soil and an anaerobic soil. They observed a half-life in these systems of approximately 10 days; no degradation was observed with an aerobic soil.

In a similar study, Kohnen, et al. (1975) found half-lives of 18.7 and 10.7 weeks with moist, aerated sterilized and unsterilized soils, respectively. Under anaerobic conditions, degradation was more rapid but there was little evolution of $^{14}CO_2$ from the labelled starting material. Sethunathan (1973) noted that all BHC's were readily degraded under anaerobic conditions although the β- and δ- isomers were more stable than either the α- or lindane forms. Suzuki, et al. (1975), who also investigated a number of BHC isomers on soil, found that the relative persistences were β- > δ- > α- > lindane.

In an investigation particularly relevant to freshwater ecosystems, Hamelink and Waybrant (1976) reported that the observed rate of disappearance of lindane corresponded to a half-life of 18-26 weeks but included removal via sedimentation as part of "degradation". Oloffs, et al. (1972, 1973), using water from the Fraser River, observed that no degradation occurred within 12 weeks when no sediment was present. However, when sediments were included, all material became associated with that compartment. Furthermore the data indicated that lindane degraded with a half-life of approximately 2 weeks. Yamamoto, et al. (1980), in an actual field situation (river), found a sinusoidal seasonal fluctuation in the concentrations with a rate corresponding to a half-life of approximately 36 weeks.

Metabolism

Most information about metabolic routes and products pertains to mammalian systems. Although some data exist for soil microorganisms there is little for aquatic organisms. In rats, Kurihara, et al. (1979) have shown that lindane induces liver microsomal activity, particularly P-450 cytochrome which appears to be essential to the degradation process. A number of metabolic mechanisms have been suggested for the rat with Stein, et al. (1977) concluding that oxygenation leads to chlorinated phenols.Other mechanisms include dehydrochlorination which yields pentachlorocyclohexene; dehydrogenation producing hexachlorocyclohexene; and dechlorination forming tetrachlorocyclohexenes (Chadwick, et al., 1981; Stein, et al., 1980; Weisburger, 1978). Two reviews of these processes have suggested that (Portig, 1983; Engst, et al., 1977 and 1979) the products of these primary reactions are, of course, subject to subsequent metabolism/reaction. As such chlorinated benzenes (Seidler, et al., 1975), chlorinated phenols (Tanaka, et al., 1977; Freal and Chadwick, 1973) and chlorocyclohexenols (Engst, et al., 1976), have been reported as final degradation products. Chlorobenzenes and chlorophenols would seem to be the major, stable metabolites of lindane in rabbits (Karapally, et al., 1973). In a case of accidental human poisoning, the identified metabolites were di- and trichlorophenols (Starr and Clifford, 1972).

Similar reactions and products for lindane degradation are reported in microbial soil systems. It would appear that the reductive processes proceed further here than in the mammalian systems since the chlorinated benzenes are more frequently reported (Jagnow, et al., 1977 and Kohnen, et al., 1975). In addition to these compounds, Haider (1979) also reported the production of penta- and tetrachlorocyclohexenes under anaerobic conditions.

Other BHC isomers also undergo metabolic degradation. Freal and Chadwick (1973) established that the rates of degradation were in the order γ- > δ- > α- > β-BHC. Koransky, et al. (1975) showed that the reaction of the α- isomer proceeded similarly to lindane in rats. Tanaka, et al. (1977) found that in rat livers, the ease of formation of the trichlorophenols was δ- > α- > γ- > β-BHC.

It should be noted that in target organisms such as terrestrial insects, a resistance to lindane (and presumably to other BHC isomers) can be developed which is associated with the metabolism to pentachlorocyclohexene (Sternburg and Kearns, 1956). The same observations are reported by Brown (1971) who suggested that genetically improved metabolism and central nervous system tolerance were responsible. He also noted that a cross resistance to lindane was developed in flies following their exposure to the chlorinated cyclopentadiene insecticides.

Exposure in the Great Lakes

Lindane and the other BHC isomers have been repeatedly reported for the air, water, sediment and biota compartments of the Great Lakes. Eisenreich, et al. (1981), in their review of atmospheric data, estimated that mean air and rainfall concentrations for lindane in the region were 0.002 $\mu g/m^3$ and 0.005 $\mu g/L$, respectively with α-BHC it was 0.0003 $\mu g/m^3$ and 0.015 $\mu g/L$, respectively. These values resulted in total BHC deposition estimates of 1-3 t/yr. and 4-16 t/yr. for each of the Great Lakes via these two media. Seiber, et al. (1983) reported mean values for the air of U.S. cities to be 0.0011 μg α-BHC/m^3 and 0.0009 μg lindane/m^3 which are in accord with the total air estimates of Eisenreich, et al. (1981). Galloway, et al. (1980) similarly determined air and rainfall concentrations for α-BHC to be 0.002 $\mu g/m^3$ and 0.010 $\mu g/L$, respectively.

Other sources of lindane include direct discharges from industrial and sewage treatment sites. The Niagara River Toxics Committee (1984) reported lindane in effluents, leachates and waste dumps from a variety of locations in the Niagara area with obvious connotation to the raw water supply of communities on the lower Niagara River and Lake Ontario. Roebeck, et al. (1965), in a study of treatment processes, found that conventional, ozone or permanganate treatments were ineffective at removing more than 10% of the lindane present in raw water.

A recent review of toxic contaminants in Lake Ontario (Strachan and Edwards, 1984), included data from a variety of sources and indicated annual loadings from the Niagara River of 2.3 tonnes of dissolved lindane and a relatively minor amount via the suspended load. Waters for the lake itself were variously reported as 0.0001-0.006 µg/L and for the sediments 0.004-0.06 µg/g. Data for fishes were far fewer and ranged from 0.01-0.4 µg/g. In another study, Canonne and Mamarbachi (1975) found lindane concentrations of 0.5-3.7 µg/g in approximately 25% of sediment samples from the lower St. Lawrence River. In Herring gulls the β- isomer was found at a mean level of 35 µg/g and 0.078 µg/g for gull lipid and gull eggs, respectively (Norstrom, et al., 1978; Gilman, et al., 1977).

Other lakes in the system do not have as much data as exists for Lake Ontario. A summary of some of these data is presented in Table 2.3.2.

For aquatic life, it is apparent that there is a continuing exposure from the water at the nanogram/litre level and that this is probably widespread in the system. The field significance of this exposure has not been established, but as with other substances, the assumption is made that laboratory findings are appropriate surrogates to the field.

It is also apparent from the values in Table 2.3.2 that there is a possible human daily intake of BHC isomers of 0.01 µg in 2L drinking water and another possible 0.03 µg via inhalation (0.5L tidal volume, 29 breaths/minute) which are of considerably lower significance when compared with even a single meal of a fish with a level of 0.05 µg/g (a 120 g portion of which would contain 6 µg). Various other human food sources have been examined for their BHC isomer content. Albert (1983) found mainly β-BHC in dairy products and potatoes contained mainly the lindane form in samples collected in Mexico during 1980. In France, Deschamps and Hascoet (1983) reported human BHC food intake to be mainly via dairy and cereal products. Cereal crops (oats) are reported to take up lindane from different soils (Fuhremann and Lichtenstein, 1980) and other food sources have also been observed to be contaminated with isomers of BHC (IARC, 1980). Indeed, food is described as the major exposure medium for humans. Intake by humans in the Great Lakes, noted above when compared with the Acceptable Daily Intake of 700 µg for a 70 kg human (FAO/WHO, 1978) suggest that with any acceptable proportion of this value allocated to drinking water, the limit would be well above the objective recommended in this document.

A review by Kutz, et al. (1974) identified 0.6 µg/g of the β- isomer (at 95% of total BHC) in human adipose tissue; Savage, et al. (1973) reported 38 µg/g in human breast milk, from a Colorado study; Polishuk, et al., (1970) reported adipose tissue from non-pregnant women contained 0.42 µg/g and that from pregnant women was 0.14 µg/g ; blood levels for the β- form were approximately 10 times those of lindane.

Table 2.3.2: Lindane and αBHC Concentrations in Great Lakes Other than Lake Ontario

SAMPLE	LOCATION	(YEAR)	CONCENTRATION µg/L αBHC	or µg/g Lindane	REFERENCE
WATER	St. Marys River	(1967)		0.003	Lichtenberg, et al., 1980
	Lake Superior	(1973-5)		0.002-0.008	Strachan and Glass, 1978
	Lake Huron/Superior	(1975)		<0.005	Glooschencko, et al., 1976
	Grand River	(1976)	0.0019	0.00067	IJC, 1978
	Saginaw Bay/ Detroit River	(1967)		0.002, 0.007	Lichtenberg, et al., 1970
DRINKING WATER	Various Ontario locations	(1981)	0.0073	0.0025	Williams, et al., 1982
STP EFFLUENTS	Detroit River	(1973-5)		0.003-0.054	IJC, 1978
	Other U.S.	(1973)		0.001-0.016	IJC, 1978
RAIN	Various Ontario locations	(1977)	0.012	0.005	Strachan and Huneault, 1979
AIR (mg/m³)	Various U.S. locations	(unknown)	0.003	0.002	Eisenreich, et al., 1981
SEDIMENT	Grand River	(1976)	0.016	0.004	IJC, 1978
Chub	Lake Superior	(1974)		0.10	IJC, 1977
Burbot	Lake Superior	(1974)		0.05	IJC, 1977
Sculpin	Lake Superior	(1974)		0.01	IJC, 1977
Lake Trout	Lake Superior	(1974-6)	0.005-0.009	0.01-0.12	IJC, 1978
Chub	Lake Huron	(1974)		0.04	IJC, 1977
Burbot	Lake Huron	(1974)		0.02	IJC, 1977
Sculpin	Lake Huron	(1974)		0.03	IJC, 1977
Minnows	Lake Huron	(1977)		0.002-0.007	IJC, 1978
Various fish	U.S. Great Lakes	(1967-8)		0.01-0.36	Henderson, et al., 1969
Starlings	U.S. Great Lakes	(1967-8)		(0.01)	Martin, 1969

Aquatic Toxicity

Compared with its effects on insects and fishes, lindane is relatively non-toxic to aquatic plants. Among the more sensitive responses observed, Jeanne (1979) noted inhibition of cell division in unicellular marine algae at 5 mg/L; morphological changes were also observed. Qualitatively similar results were found by Borghi, et al. (1973) and Escoubet (1978).

Comparable studies with freshwater algae do not appear to have been done for species resident in the Great Lakes. Related species and their inhibiting concentration levels which have been reported include Ankistrodesmus braunii and Anacystis nidulans at approximately 10 mg/L (Kopecek, et al., 1975) and Chlorella at > 0.3 mg/L (Hansen, 1979). Macrophyte (Elodea densa) membrane potentials were also adversely affected after a 10 hour exposure at 10 mg/L to the extent that the leaves were no longer sensitive to light (Schefezik and Simonis, 1980).

Lethal effects of lindane have been observed with Gammarus pulex (Abel, 1980) in which the influence of both the exposure time and the concentration were investigated. At the lowest concentration reported, 10 µg/L, the median survival time was 116 hours. However, death continued to be observed for up to three weeks after placing the animals in clean water. At 100 µg/L, data from the curves presented indicated that an increase of exposure time from 100 to 1000 minutes resulted in a decrease in the median survival time of 1000 to 100 hours.

In other studies with invertebrates, Bluzat and Seuge (1979) reported 48-h LC_{50}'s for Gammarus, Cloeon, Chaoborus and Lymnea of 30, 92, 8 and 7300 µg/L, respectively. Lymnea showed decreased growth at 2 mg/L and a decrease in fecundity (36%), egg fertility and increased embryogenesis (12%) at 1 mg/L. Macek, et al. (1976), in a series of determinations in which various water quality parameters were recorded as well, observed 48-h LC_{50}'s for Chironomus tentans, Daphnia magna and Gammarus fasciatus of 207, 485 and 39 µg/L, respectively. This study also focussed on survival and reproductive effects of the three animals. The lowest concentrations, resulting in adverse reproduction were 7.3, 19 and 8.6 µg/L, respectively. Combination of the arthropod lethality data with that of the reproductive responses gives application factors of 0.035, 0.039 and 0.22, respectively, for the three species in Macek, et al. (1976) and 0.14 for Lymnea from Bluzat and Seuge (1979). The geometric mean of these is 0.081. Other studies indicating sensitivity of aquatic invertebrates to lindane include those with the stonefly nymph (Pteronarcys californica) which had a 48-h LC_{50} of 1 µg/L (Cope, 1965) or 4.5 µg/L (Sanders and Cope, 1968) and the scud (Gammarus fasciatus) with a 96-h LC_{50} of 10 µg/L (Sanders, 1972). Another sensitive species is the mayfly nymph, a genus for which there are many representative species in the Great Lakes basin. The nymph Baetis rhodani showed reduced survival effect at 0.1 µg/L (Harper, et al., 1977). In their quarry system, which included zooplankton and fish, Canton, et al., (1975) concluded that a system no-effect level was 0.05 µg/L.

In a study with α-BHC, Canton, et al. (1975) observed a 48-h LC_{50} for Daphnia magna of 800 µg/L, approximately 1.7 times that of the lindane isomer (Macek, et al., 1976).

Studies generally indicate that fish are less sensitive to BHC-isomers than the invertebrates. A number of investigations on lindane, however, indicate that fish and invertebrates may be equally at risk, at least from lindane. Rainbow trout (Salmo gairdneri) exposed to lindane at

concentrations as low as 1 µg/L showed adverse effects on protein utilization (Mendiola, et al., 1981). In another study, Zambriborshch and Bui (1976) using paradise fish fry (Macropodus opercularis), observed lethality of concentrations of 3-18 µg lindane/L during 24 hour exposure. Brown trout 96-h LC_{50}'s of 2 µg/L have also been reported (Macek and McAllister, 1970).

Data cited in the U.S. Ambient Water Quality Criteria document (EPA, 1980), indicate that the lindane isomer is only slightly more toxic (96-h LC_{50} of 1-2 µg/L) to trout species than are the α- (2-4 µg/L) and δ- (4-8 µg/L) isomers, although considerably more so than the β- form (30-60 µg/L).

There are few studies related to other aquatic organisms, at least where effects occur at concentration levels comparable to those reported for sensitive invertebrates and fishes. Fifty percent mallard mortality was observed (Tucker and Crabtree, 1970) when they were fed a diet including lindane at 30 µg/g over thirty days. The myofilaments of frog muscle (Publicover, et al., 1979) were reported to be damaged at 1.5 mg/L.

Mammalian Effects

The effects of lindane on mammals has been reviewed by the IJC's Committee on the Assessment of Human Health Effects of Great Lakes Water Quality (IJC, 1981) and by the World Health Organization (IARC, 1980). In the former's mammalian toxicology profiles, information is presented to the effect that lindane and other isomers are acutely toxic to rodents at 60-360 µg/g (LD_{50}'s) while the estimated LD_{50} for humans was 125 µg/g for lindane and 400 µg/g for total BHC (IARC, 1980). Lindane and α-BHC are described as liver carcinogens for mice but β-BHC is considered only as a suspected carcinogen (Allen, et al., 1979; IARC, 1980). Both α-BHC and lindane have been found to be weakly mutagenic with hamster cells and with bacteria (NIOSH, 1980).

Long-term (32 weeks) lindane feeding studies with dogs caused liver enlargement at 200 µg/g in the diet but demonstrated no other histopathological effects (Sternberg, 1979). Two-year investigations using rats demonstrated liver and kidney damage at 50-100 µg/g in the diet and estimated a no-effect level of 25 µg/g (Herbst, 1973).

Other adverse effects found with mammalian species include: central nervous system effects with humans (Allen, et al., 1979; IARC, 1980; WHO, 1981) as well as biochemical effects (Solomon, et al., 1977); reduced fetal development in dogs fed at dietary levels of 8-15 µg/g (WHO, 1981); and reduced conception rates in mice at 8 µg/g (WHO, 1981). Studies with the rabbit (Sternberg, 1979), rat and mouse (WHO, 1981) did not show any teratogenic effects at levels of 15-60 µg/g.

Current Control Measures

A number of regulations and guidelines exist for lindane. These include: U.S. EPA's criterion of 0.08 µg/L of lindane for the protection of freshwater aquatic life; EPA's 4 µg/L standard for the provision of a suitable domestic water supply; and, a Canadian drinking water objective level of 0.0001 µg/L with a maximum acceptable concentration of 4 µg/L.

In addition to the above, there is a U.S. FDA guideline of 0.3 mg/kg in edible fish tissue. There is also an assessment in the EPA criteria document (EPA, 1980) which indicates that protection will be afforded to humans against carcinogenic effects at a risk level of $1:10^6$ with 0.018 µg lindane/L (α-BHC at 0.009 µg/L and other isomers at intermediate levels) and to aquatic organisms at 0.060 µg lindane/L (α-BHC at 0.030 µg/L). A maximum occupational exposure limit of 0.5 mg/m^3 also exists (U.S. OSHA, 1976).

Finally, the 1978 Great Lakes Water Quality Agreement (1978) presently includes an ecosystem objective of 0.01 µg lindane/L (based on an invertebrate application factor of 0.01 for aquatic invertebrates and a 96-h LC_{50} of 1 µg/L for the stonefly). It also includes an objective of 0.3 µg/g in edible portions of fish (based on the above U.S. FDA guideline). These are to provide protection for aquatic organisms and consumers of fish, respectively.

Summary and Recommendation

The most sensitive responses related to the freshwater aquatic ecosystem would appear to be those of the invertebrates and fishes. Fishes have shown lethal effects at 1-2 µg/L while invertebrates demonstrate sublethal effects with chronic exposures at the same levels. These latter effect levels, together with the 0.2 safety factor used with persistent toxic substances in the Great Lakes (IJC, 1975), indicate an objective level of 0.2 µg/L. The freshwater fish lethality data, however, are much too close to this level and are above the 0.05 µg/L "safe" level estimated in a field situation. It is also above a sub-lethal effect level found for a relevant invertebrate. A more protective level is therefore indicated.

As a consequence of the above, it is recommended that the existing 0.01 µg/L objective for lindane be changed to 0.02 µg/L - a level obtained by consideration of the lowest invertebrate 96-h LC_{50}, 1 µg/L, the application factor of 0.081 for sensitive invertebrates and a further safety factor of 0.2 used with persistent toxic substances (IJC, 1975). The objective for edible portions of fish should remain unchanged. It is further recommended that, because of the possible conversion of lindane to other BHC isomers, both objective levels should apply to the total of the isomer forms.

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2.4 TOXAPHENE

EXISTING OBJECTIVE

The concentration of toxaphene in water should not exceed 0.008 micrograms per liter for protection of aquatic life.

RECOMMENDED OBJECTIVE

The concentration of toxaphene in water should not exceed 0.0002 micrograms per liter for the protection of human consumers of fishes.

RATIONALE

Introduction

Toxaphene first became commercially available in 1948 under the trade name "Hercules 3956" and has since been used in several formulations (emulsifiable concentrate, wettable powder, dusts and granular baits). Toxaphene is produced by the chlorination of camphene, resulting in a mixture or at least 177 separate components with a total chlorine content of 67 to 69% (Holmstead, et al., 1974). It is frequently mixed with methyl parathion or other pesticides to improve its effectiveness. It was the most heavily used pesticide in the U.S. during the 1960s and 1970s, annual applications totalling many millions of kilograms (Pollock and Kilgore, 1978; Ribick, et al.,1982). It has been employed against insect pests of cotton, tobacco, forests, turf, ornamental plants, grains, vegetables and livestock, mostly in the southern U.S. and California. When DDT was banned in 1971, toxaphene replaced many of DDT's former uses. In 1976 it was close behind methyl parathion as the most heavily used insecticide in the "delta states" of Louisiana, Arkansas and Mississippi (4.6 million kilograms) and the sixth most heavily used in the corn belt (0.2 million kilograms) (Schmitt and Winger, 1980). A total of 0.7 million kilograms was applied to a wide range of major agricultural crops in 12 north central states in 1978 (Acie and Park, 1981). Use in California in the 1970's averaged 0.8 million kilograms per year (Cohen, et al., 1982). Toxaphene's relatively low toxicity to honey bees compared to many other insecticides has favoured its use agriculturally (Eckert, 1949). Only very small quantities of toxaphene have ever been used agriculturally in Canada (Department of National Health and Welfare Canada, 1977). It was also used in the 1950's and early 60's by fisheries personnel in several U.S. states and Canadian provinces to remove unwanted fish from lakes and ponds. This use was discontinued or prohibited when its greater than expected persistence in some lakes was fully recognized.

Canadian and U.S. Use Regulation

The Canadian registration for all pesticidal uses of toxaphene was revoked in October, 1980, except for a minor use on hogs for lice by veterinarians. The U.S. EPA cancelled toxaphene's registration for

all uses in November, 1982, except as a treatment for scabies on cattle and sheep and emergency use for armyworms, cutworms and grasshoppers on cotton, corn and small grains. Some existing stocks could be sold and used according to label specifications through 1986 and all other stocks through 1983. However, Nor-Am Agricultural Products Inc., the U.S. manufacturer producing the largest quantity of toxaphene at that time, discontinued production in 1982 and their estimate of use that year (4.1×10^6 kilograms) indicates disposal of nearly all existing stocks before 1983 (personal communication of Robert Hitch, U.S. EPA, Washington, D.C. to Larry Fink, U.S. EPA, Chicago, Illinois).

Properties and Environmental Quantitation

The technical grade product is an amber, waxy solid with a vapor pressure of 0.17 to 0.4 mm Hg at 25°C, a melting point range of 65 to 90°C, and a mild terpene odour. Its' approximate general empirical formula is $C_{10}H_{10}Cl_8$ (mw 414). It is soluble in water to 37µg/L at ambient temperature (Lee, et al., 1968) and is slightly soluble in alcohols. It is highly soluble in organic solvents such as kerosene, acetone, benzene, chloroform, xylene and toluene. It is dechlorinated photolytically and by heat above 120°C, its breakdown being accelerated by alkaline conditions and by iron catalysis. Most of the individual components of toxaphene have not been precisely identified.

Capillary gas chromatography (GC), sometimes in combination with mass spectrometry (MS) is the most frequently used analytical method for characterization and quantitation of toxaphene in biological samples (Ribick, et al., 1982). A typical toxaphene gas chromatogram exhibits a large number of peaks, a few of which are selected to distinguish toxaphene from other possible environmental co-contaminants. The identification and quantitation of toxaphene in water and fish tissues is complicated by changes in the numbers and relative sizes of constituent peaks because of their differing rates of sorption, volatilization and degradation in the environment. The compositional changes that occur in the field probably also mean that field toxicity is to some unknown extent different from toxicity determined in laboratory tests using relatively unaltered technical grade toxaphene. Using mice, houseflies and goldfish, Turner, et al. (1977) have demonstrated that there are substantially different toxicities for a few identified components. Changes in environmental sample chromatograms as compared to reference standard chromatograms have led some analysts to refer to their values as "toxaphene-like" substances, although the prevailing uncertainty in identification using the latest analytical techniques is small. The lower limit of detection of toxaphene by several GC detection methods is about 5 to 10 ng (Durkin, et al., 1979); concentrations have been quantitatively measured in fish tissues down to 0.1 µg/g (Ribick, et al., 1982).

Presence in the Atmosphere

Sharply elevated atmospheric levels of toxaphene have been reported in the immediate vicinity of intense applications (Stanley, et al., 1971), but long range transport over several hundred kilometers was not directly demonstrated until the mid 1970's (Bidleman and Olney, 1975). Indeed, mean levels of toxaphene in western North Atlantic air, presumably carried from cotton growing areas in the southern U.S., were more than ten times higher than those of other pesticides reported in the marine atmosphere (Bidleman and Olney, 1975). Vaporization has been shown to be the major source of residue loss from foliage with essentially no short-term loss attributable to biological or chemical degradation (Seiber, et al., 1979). Shifts in atmospheric analysis profiles therefore primarily reflect differences in the volatility of individual components.

Rice, et al. (1984) monitored atmospheric levels of toxaphene in the summer and fall of 1981 at four locations between Greenville, Mississippi and northern Lake Michigan. Evidence indicating the cotton belt as a source of toxaphene in Lake Michigan included: a decrease in the number of GC chromatogram toxaphene peaks occur when sampled from south to north; a reduction in concentrations (7.39 ng/m^3 in Greenville, 1.18 ng/m^3 in St. Louis, 0.27 ng/m^3 in Michigan) from south to north; corresponding temporal concentration patterns (all higher in summer); and a net south to north wind flow pattern. The authors estimated a total toxaphene flux to Lake Michigan of 3,360 to 6,720 kg in 1981. Swain, et al. (in press) measured approximately 30 ng/L of toxaphene in precipitation collected during several months of 1980 at five stations around Lake Huron. Another investigation of persistent organic chemicals in rainfall to Lake Superior in 1983 and 1984 found no toxaphene, even though several other chlorinated pesticides, PCB and HCB, were detected in measurable quantities and it was detected in spiked samples (Strachan, 1985).

Presence in Biota

Toxaphene has been observed frequently in tissues of birds and fish both near to and distant from primary use sites: shore birds and gulls in Texas (White, et al., 1980, 1985); eagles (Wiemeyer, et al., 1984); fish-eating birds (Ohlendorf, et al., 1978) across the U.S.; ducks in California (Ohlendorf and Miller, 1984), and Maine (Szaro, et al., 1979); brown pelicans in Louisiana (Blus, et al., 1975); Canadian east-coast marine fish (Musial and Uthe, 1983); various fish species in Alabama (Grzenda and Nicholson, 1965), the Colorado River (Johnson and Lew, 1970), and California (Keith and Hunt, 1966). A few instances of elevated or unusual bird mortalities appear to be associated with agricultural or fish-control toxaphene applications (Keith, 1966). An extensive fish kill in rice field drainage canals followed aerial spraying of toxaphene to control grasshoppers in Texas (Ginn and Fisher, 1974). Plumb and Richburg (1977) reported a continuous over-winter mortality in

two Alabama ponds containing catfish with high blood serum levels of toxaphene and endrin.

Presence in the Great Lakes

It was not until the mid 1970's that analysis of fish from Lakes Michigan, Huron and Superior first indicated the existence of toxaphene in the Great Lakes. Its presence was somewhat surprising because relatively little had been used for pest control in this geographic region. Toxaphene residue levels measured in Great Lakes fish through 1981 have been summarized by Rice and Evans (1984). They indicate an increase in residues through the 1970's and higher levels in Michigan fish than in those from the other lakes. Like other chlorinated hydrocarbon pesticides, toxaphene is lipophilic and tends to reach maximum levels in the oldest and fattest fish at the top of the food chain such as lake trout. Concentrations in this species have generally ranged between 1 and 10 µg/g in recently published analyses (Schmitt, et al., 1983; Hesselberg, in Rice and Evans, 1984; Canada Dept. of Fisheries and Oceans, 1982). Schmitt, et al. (1985) reported that toxaphene residues seemed to have plateaued nationally in U.S. freshwater fish collected in 1980 and 1981, even though it was more widely distributed than in previous surveys. Residues in Great Lakes fish, especially those from Lakes Michigan and Superior, generally appeared 2 to 5 µg/g lower than the 5 to 10 µg/g commonly observed during the 1970's. Adult lake trout collected from Lake Huron near Rockport, Michigan in 1984 contained 2.2 µg/g toxaphene; bloater chubs collected from Lake Michigan near Saugatuck, Michigan in 1982 contained 1.6 µg/g, while those collected in the same area in the fall of 1984 contained 2.2 µg/g (personal communication, Dr. Robert Hesselberg, U.S. Fish and Wildlife Service, Great Lakes Fishery Laboratory, Ann Arbor, Michigan). All reported values are for concentrations in whole fish, which are probably somewhat higher than edible tissue concentrations. Clark, et al. (1984) reported "apparent toxaphene" residues below 0.5 µg/g in coho salmon fillets from Lakes Erie and Superior, and nearly 2 µg/g from Lakes Michigan and Huron. "Toxaphene- like" residues have been measured up to 26 µg/g in fillets of lake trout from the mouth of Saginaw Bay (Swain, et al., in press).

Great Lakes water determinations for toxaphene are very rare. Samples collected in 1980 from 5 stations in Lake Huron ranged from 1.2 to 2.1 ng/L and averaged 1.6 ng/L (Swain, et al., in press). The U.S. EPA laboratory at Grosse Ile, MI, has also measured "toxaphene-like" residues in Siskiwit Lake on Isle Royale in Lake Superior at 2.2 ng/L and in Lake Superior adjacent to Isle Royale at 1.0 ng/L. While the analysts still refer to these as "toxaphene like" materials, they are quite certain that the observed residues are derived from chlorinated camphene (personal communication, Dr. Mike Mullin, U.S. EPA, Grosse Ile, MI). Five composites of lake trout from Siskiwit Lake averaged 4.2 µg/g and a cross-check of these analyses by the U.S. Federal and Wildlife Service laboratory in Columbia, Missouri, measured 3.2 µg/g. Toxaphene has

been measured in the water at several additional sites around Lake Superior since 1982 (Dr. Steve Eisenreich, University of Minnesota, Minneapolis, personal communication). Water concentrations range from 1 to 4 ng/L with the higher values being present at the western end of the lake. No water concentration determinations are known to exist for the other Great Lakes.

Haseltine, et al. (1981) reported an accumulation of toxaphene in the eggs of waterfowl nesting on islands in northern Lake Michigan. Fifty percent or more of the eggs of 206 fish-eating mergansers analysed in 1977 and 1978 contained means of 0.14 and 0.27 µg/g toxaphene respectively, while most of the eggs of surface-feeding ducks had little or no detectable residues. Merganser eggs exhibited a slight degree of shell thinning attributable to DDE residues, but no other adverse effects were observed (Heinz, et al., 1983). Snakes and their terrestrial food items on these islands contained no detectable residues (Heinz, et al., 1980).

Persistence in Water

Toxaphene applications to lakes related to fisheries management practices have provided substantial amounts of aquatic fate and effects field data. Reports are available on the treatment of water bodies in at least a dozen states and three provinces. Most of these studies investigated the diminution of toxaphene water concentrations over time as an indication of its efficacy and how soon after elimination of undesirable species the lake could be restocked. Treatment concentrations usually ranged between 5 and 200 µg of toxaphene per liter of lake water, higher concentrations being recommended for warmer, shallower and more turbid lakes (Rose, 1958). Persistence of conditions toxic to fish was highly variable, ranging from a few weeks (Mayhew, 1959) to greater than five years in Miller Lake, Oregon (Terriere, et al., 1966). Water concentrations typically dropped rapidly within a day or two after application due to sorption to suspended particulates or to the sediment (Veith and Lee, 1971) and then diminished much more slowly for an indefinite period (Veith and Lee, Kallman, et al., 1962). The chemical persisted longest in hypolimnetic areas of the most oligotrophic lakes (Stringer and McMynn, 1960; Terriere, et al., 1966) but was detected at 1-4 µg/L up to 10 years after it was applied to shallow eutrophic lakes in Wisconsin (Johnson, et al., 1966). Various studies (Chandurkar and Matsumura, 1979; Isensee, et al., 1979; Hughes, et al., 1970) have demonstrated a biological capability to metabolize or degrade toxaphene both aerobically and anaerobically. Quantitative data on degradation in water are lacking although it is obviously very slow under some conditions. Nash and Woolson (1967) estimated the half-life of toxaphene to be 11 years in soil. Veith and Lee (1971) observed an initial half-life in the sediments of two Wisconsin lakes of 20 days which subsequently increased to 120 days. Toxaphene is not readily desorbed back into water from contaminated sediments (Veith and Lee, 1971) but is likely to be cycled within aquatic ecosystems through the benthos-water column food web connections (Kallman, et al., 1962; Rice and Evans, 1984).

Aquatic Organism Toxicity

Table 2.4.1 presents much of the acute toxicity information available for vertebrate and invertebrate aquatic organisms generated under diverse exposure conditions and with varied sources or stocks of animals. Several routinely measured water quality factors (e.g., dissolved oxygen concentration, hardness and/or alkalinity, pH, etc.) were omitted from the table because of space limitations. There have been only a few comparative studies of such water quality factors: Johnson and Julin (1980) found essentially no effect of a 6.5 to 8.3 pH range on the toxicity of toxaphene to channel catfish. Johnson and Julin (1980), Hooper and Grzenda (1955) and Henderson, et al. (1959) found less than a two-fold difference in toxicity to channel catfish and fathead minnows in hard (>200 mg/L) versus soft (<20 mg/L) water. Workman and Neuhold (1963) compared the toxicity of a floating oil emulsion formulation to a sinking kerosene-carrier formulation using three fish species tested in three water types varying in measured pH (7.0-8.3), temperature (8-20°C) and total dissolved solids (46-238 mg/L). While the total range in toxicity was 4 to 48 µg/L, formulation appeared to make little difference (Table 2.4.1). From these limited data and the few additional less comparable data sets in Table 2.4.1 (same organism, similar exposure conditions, different formulations) it appears that in most cases formulation does not greatly influence toxicity. Temperature, on the other hand, does appear to influence acute results, the toxicity of toxaphene being greater at higher temperatures (Cope, 1964; Mahdi, 1966; Hooper and Grzenda, 1955; Workman and Neuhold, 1963). The data in Table 2.4.1 are arranged to facilitate comparisons of LC_{50} values between exposures of equal duration of similar life stages or organisms at different temperatures.

Two other factors, exposure duration (lower LC_{50} values with longer exposures) and static versus flowing test methodology, also obviously influence acute results. Toxaphene is relatively insoluble (37 µg/L) in water (Lee, et al., 1968) and tends to sorb onto solid surfaces and particulates, especially those containing organic materials. Actual water concentrations of toxaphene are normally lower than amounts introduced into either flowing or static test systems, but are particularly lower in static tests. For example, Hall and Swineford (1981) measured an average of only 30.5% of the intended water concentrations in a series of static acute tests, while in a series of continuous-flow exposures they obtained 55.4% of the amounts intended in their test waters (Hall and Swineford, 1980). Other flow-through tests probably maintained water concentrations somewhat closer to calculated values, but most of the unmeasured values in Table 2.4.1 and 2.4.2 indicate higher levels of toxaphene than were experienced by test organisms.

TABLE 2.4.1: LABORATORY ACUTE MORTALITY DATA

Organism	Toxicant Formulation	Life Stage or Size	Temperature (°C)	Test Method (Static, Flow-Through, Renewal; Measured, Unmeasured)	Exposure Duration[1]	Endpoint	Endpoint Concentration (µg/l)	Reference
Clam, Rangia cuneata	NR[2]	35-50 mm	NR	NR,U	96 hr	LC50	460,000	Chaiyarach, et al., 1975
Clam, Daphnia magna	EC[3]	NR	12.7	S,U	24 hr	TLM	1,500	Hooper and Grzenda, 1955
	NR	1st Instar, <24 hr	19	R,U	26 hr	LC50	94	Freal and Boyd, 1967
	C[4]	NR	21.1	S,M	26 hr	EC50IM[5]	260-	Crosby, et al., 1966
	C	NR	25	S,M.	26 hr	EC50IM	1,900	Crosby, et al., 1966
	Tech.[6]	1st instar	21	S,U	48 hr	EC50IM	10	Johnson and Finley, 1980
	NR	<24 hr	23	S,U	48 hr	EC50IM	155	Bringmann and Kuhn, 1960
Cladoceran, Daphnia pulex	NR	1st instar	15.6	S,U	48 hr	EC50IM	15	Sanders and Cope, 1966
Cladoceran, Simocephalus serrulatus	NR	1st instar	15.6	S,U	48 hr	EC50IM	19	Sanders and Cope, 1966
	NR	1st instar	21.1	S,U	48 hr	EC50IM	10	Sanders and Cope, 1966
Isopod, Asellus intermedius	EC	NR	12.7	S,U	24 hr	LC50	100	Hooper and Grzenda, 1955
Amphipod, Gammarus fasciatus	EC	NR	12.7	S,U	24 hr	LC50	60	Hooper and Grzenda, 1955
	Tech.	NR	21	S,U	24 hr	LC50	82	Sanders, 1972
	EC	NR	21	S,U	24 hr	LC50	47	Sanders, 1972
	EC	NR	21	S,U	96 hr	LC50	6 & 35	Sanders, 1972
Amphipod, Gammarus lacustris	Tech.	15-20 mm	NR	S,U	7.67 hr	LT50[7]	50	MacDonald, 1962
Prawn, Palaemonetes kadiakensis	Tech.	NR site 1	24	S,U	24 hr	LC50	44	Naqvi and Ferguson, 1970
	Tech.	NR, site 2	24	S,U	24 hr	LC50	229	Naqvi and Ferguson, 1970
	Tech.	NR, site 3	24	S,U	24 hr	LC50	20.9	Naqvi and Ferguson, 1970
	Tech.	NR, site 4	24	S,U	24 hr	LC50	80.9	Naqvi and Ferguson, 1970
	Tech.	NR, site 1	20	S,U	36 hr	LC50	170	Ferguson, et al., 1965b
	Tech.	NR, site 2	20	S,U	36 hr	LC50	57.5	Ferguson, et al., 1965b
	Tech.	Late instar	21	S,U	96 hr	LC50	28	Sanders, 1972
	NR	25-31 mm	NR	NR,U	96 hr	LC50	36	Chaiyarach, et al., 1975
White River crayfish, Procambarus acutus acutus	Tech.	0.25-0.40 g: 11.8-14.6	NR	S,U	48 hr	EC50IM	60.7	Albaugh, 1972
Crayfish, Procambarus s. simulans	NR	60-70 mm	NR	NR,U	96 hr	LC50	210	Chaiyarach, et al., 1975

TABLE 2.4.1: LABORATORY ACUTE MORTALITY DATA (Continued)

Organism	Toxicant Formulation	Life Stage or Size	Temperature (°C)	Test Method (Static, Flow-Through, Renewal; Measured, Unmeasured)	Exposure Duration[1]	Endpoint	Endpoint Concentration (µg/l)	Reference
Mayfly, Epemera simulans	EC	NR	23.7	S,U	24 hr	LC50	9,500	Hooper and Grzenda, 1955
Stonefly, Pteronarcella badia	Tech.	15-20 mm	15.5	S,U	96 hr	LC50	3.0	Sanders and Cope, 1968
Stonefly, Pteronarcys californica	Tech.	30-35 mm	15.5	S,U	96 hr	LC50	2.3	Sanders and Cope, 1968
Stonefly, Claassenia sabulosa	Tech.	20-25 mm	15.5	S,U	96 hr	LC50	1.3	Sanders and Cope, 1968
Mosquito, Aedes aegypti	NR	Larva, 2nd instar	NR	S,U	<5 hr	ET50IM[9]	10	Burchfield and Storrs, 1954
	Tech.	Larva, 4th instar	NR	S,U	24 hr	LC50	375	Chandurkar, et al., 1978
	Tech.	Larva, 4th instar; site	21-23	S,U	48 hr	LC50	1,900	Klassen, et al., 1965
	Tech	Larva, 4th instar; site 2	21-23	S,U	48 hr	LC50	<81,920	Klassen, et al., 1965
	Tech.	Larva, 4th instar; site 3	21-12	S,U	48 hr	LC50	140	Klasses, et al., 1965
Crane fly, Tipula	Tech.	Larva	15	S,U	96 hr	LC50	18	Johnson and Finley, 1980
Midge Chironomus	Tech.	Larva	15	S,U	48 hr	LC50	30	Johnson and Finley, 1980
Snipe fly, Atherix	Tech.	Larva	15	S,U	96 hr	LC50	40	Johnson and Finley, 1980
Coho salmon, Oncorhynchus kisutch	Tech.	1 g	12	S,U	96 hr	LC50	8	Johnson and Finley, 1980
	Tech.	57-76 mm; 2.7-4. 1 g	20	S,U	96 hr	LC50	9.4	Katz, 1961
Chinook salmon, Oncorhynchus tshawytscha	Tech.	51-114 mm	20 C	S,U	96 hr	LC50	2.5	Katz, 1961
	Tech.	1.45-5 g NR	14.4	S,U	96 hr	LC50	1.54	Schoettger, 1970

TABLE 2.4.1: LABORATORY ACUTE MORTALITY DATA (Continued)

Organism	Toxicant Formulation	Life Stage or Size	Temperature (°C)	Test Method (Static, Flow-Through, Renewal; Measured, Unmeasured)	Exposure Duration[1]	Endpoint	Endpoint Concentration (µg/l)	Reference
Rainbow trout, *Salmo gairdneri*	NR	~1 g	7.2	S,U	96 hr	LC50	5.4	Cope, 1964
	C	NR	11.7	S,U	96 hr	LC50	8.4	Mahdi, 1966
	Tech	1.4 g	12	S,U	96 hr	LC50	10.6	Johnson and Finley, 1980
	NR	~1 g	12.8	S,U	96 hr	LC50	2.7	Cope, 1964
	Floating	21 g	12.8	S,U	96 hr	LC50	28	Workman and Neuhold, 1963
	Sinking	21 g	12.8	S,U	96 hr	LC50	23	Workman and Neuhold, 1963
	Tech.	0.6-1.7 g	15	S,U	96 hr	LC50	11	Macek and McAllister, 1970
	NR	~1 g	18.3	S,U	96 hr	LC50	1.8	Cope, 1964
Rainbow trout (Donaldson trout), *Salmo gairdneri*	Tech.	51-79 mm;	20	S,U	96 hr	LC50	8.4	Katz, 1961
Brown trout, *Salmo trutta*	Tech.	1.7 g	12	S,&	96 hr	LC50	3.1	Johnson and Finley, 1980
Brook trout, *Salvelinus fontanalis*	NR	Yearling; 133 g; 231 mm	10	F,M	96 hr	LC50	10.8	Mayer, *et al.*, 1975
Brook Trout, *Salvelinus fontinalis*	NR	Yearling; 133 g; 231 mm	10	F,M	5-8 day	LC50	7.5-4.9	Mayer, *et al.*, 1975
Stoneroller, *Campostoma anomaium*	C	NR	11.7	S,U	96 hr	LC50	14	Mahdi, 1966
	C	NR	17.2	S,U	96 hr	LC50	7	Mahdi, 1966
	C	NR	22.7	S,U	96 hr	LC50	32	Mahdi, 1966
Goldfish *Carassius auratus*	NR	NR	20	S,M	24 hr	LC50	20	Turner, *et al.*, 1977
	Floating (10% a.i.)	4.2 g	8.3 test water 4	S,U	96 hr	LC50	26	Workman and Neuhold, 1963
	Sinking (62.6% a.i.)	4.2 g	8.3 test water 4	S,U	96 hr	LC50	44	Workman and Neuhold, 1963
	C	NR	11.7	S,U	96 hr	LC50	94	Mahdi, 1966
	C	NR	17.2	S,U	96 hr	LC50	28	Mahdi, 1966
	Tech.	1 g	18	S,U	96 hr	LC50	14	Johnson and Finley, 1980
	Floating	4.2 g	20 test water 1	S,U	96 hr	LC50	4	Workman and Neuhold, 1963
	Sinking	4.2 g	20 test water 1	S,U	96 hr	LC50	9	Workman and Neuhold, 1963
	Floating	4.2 g	20 test water 2	S,U	96 hr	LC50	28	Workman and Neuhold, 1963

TABLE 2.4.1: LABORATORY ACUTE MORTALITY DATA (Continued)

Organism	Toxicant Formulation	Life Stage or Size	Temperature (°C)	Test Method (Static, Flow-Through, Renewal; Measured, Unmeasured)	Exposure Duration[1]	Endpoint	Endpoint Concentration (µg/l)	Reference
Goldfish, *Carassius auratus*	Sinking	4.2 g	20 test water 2	S,U	96 hr	LC50	16	Workman and Neuhold, 1963
	Floating	4.2 g	20 test water 3	S,U	96 hr	LC50	7	Workman and Neuhold, 1963
	Sinking	4.2 g	20 test water 3	S,U	96 hr	LC50	9	Workman and Neuhold, 1963
	C	NR	22.7	S,U	96 hr	LC50	50	Mahdi, 1966
	NR	6 cm	25	F,U	96 hr	LC50	11	Warner, *et al.*, 1966
Carp, *Cyprinus carpio*	Tech.	0.6 g	18	S,U	96 hr	LC50	3.7	Johnson and Finley, 1980
Golden shiner *Notemigonus crysoleucas*	C	NR	11.7	S,U	24 hr	LC50	12.5	Mahdi, 1966
	C	NR	17.2	S,U	72 hr	LC50	6.2	Mahdi, 1966
	C	NR	22.7	S,U	96 hr	LC50	6	Mahdi, 1966
Bluntnose minnow, *Pimephales notatus*	C	NR	11.7	S,U	96 hr	LC50	30	Mahdi, 1966
	C	NR	17.2	S,U	96 hr	LC50	8.8	Mahdi, 1966
	C	NR	22.7	S,U	96 hr	LC50	6.3	Mahdi, 1966
Fathead minnow, *Pimephales promelas*	EC	NR	10	S,U	24 hr	LC50	36	Hooper and Grzenda, 1955
	EC	NR	23.8	S,U	24 hr	LC50	5.7	Hooper and Grzenda, 1955
	NR	3-3.5 cm	NR	S,U	48 hr	LC50	77.55	Chandurkar, *et al.*, 1978
	Tech.	0.6-1.7 g	18	S,U	96 hr	LC50	14	Macek and McAllister, 1970
	EC	0.5-1.5 g	20	S,U	96 hr	LC50	20 & 23	Johnson and Julin, 1980
	Tech.	1.1 g	20	S,U	96 hr	LC50	18	Johnson and Finley, 1980
	Tech.	0.5-1.5 g	20	F,M	96 hr	LC50	7.0	Johnson and Julin, 1980
	Tech.	0.5-1.5 g	25	F,M	96 hr	LC50	7.2	Johnson and Julin, 1980
	NR	30 day; 0.32 g; 30 mm	25	F,M	96 hr	LC50	7.2	Mayer, *et al.*, 1977
	NR	30 day; 0.32 g; 30 mm	25	F,M.	5-10 day	LC50	6.4-4.8	Mayer, *et al.*, 1977
	Tech.	0.5-1.5 g	20	F,U	8-24 day	LC50	4.3-2.6	Johnson and Julin, 1980

TABLE 2.4.1: LABORATORY ACUTE MORTALITY DATA (Continued)

Organism	Toxicant Formulation	Life Stage or Size	Temperature (°C)	Test Method (Static, Flow-Through, Renewal; Measured, Unmeasured)	Exposure Duration[1]	Endpoint	Endpoint Concentration (µg/l)	Reference
Black Bullhead, Ictalurus melas	Tech.	Fingerling Site 1	20	S,U	36 hr	LC50	12.5	Ferguson, et al., 1965a
	Tech.	Fingerling Site 2	20	S,U	36 hr	LC50	50	Ferguson, et al., 1965a
	Tech.	Fingerling site 3	20	S,U	36 hr	LC50	3.75	Ferguson, et al., 1965a
	Tech.	Fingerling site 4	20	S,U	36 hr	LC50	22.5	Ferguson, et al., 1965a
	C	NR	11.7	S,U	96 hr	LC50	25	Mahdi, 1966
	C	NR	17.2	S,U	96 hr	LC50	2.7	Mahdi, 1966
	Tech.	0.6-1.7 g	18.0	S,U	96 hr	LC50	5	Macek and McAllister, 1970
	C	NR	22.7	S,U	96 hr	LC50	1.8	Mahdi, 1966
	Tech.	0.9 g	24	S,U	96 hr	LC50	5.8	Johnson and Finley, 1980
Channel catfish,	Tech.	Yolk sac	25	S,M	96 hr	LC50	8.0	Johnson and Julin, 1980
	NR	10.0 g	26	S,U	24 hr	LC50	20	Carter and Graves, 1972
	Tech.	Fingerling;	15	F,M.	96 hr	LC50	4.7	Johnson and Julin, 1980
	Tech.	1.5 g	18	S,U	96 hr	LC50	13.1	Johnson and Finley, 1980
	Tech.	4 g	20	F,M	96 hr	LC50	5.5	Johnson and Julin, 1980
	Tech.	Fingerling;	20	F,M	96 hr	LC50	2.7-7.9	Johnson and Julin, 1980
	Tech.	4 g	20	F,M	8-29 day	LC50	4.4-1.9	Johnson and Julin, 1980
	Tech.	Fingerling;	25	S,M	96 hr	LC50	0.8 & 2.8	Johnson and Julin, 1980
Mosquitofish, Gambusia affinis	NR	3.0-4.0 cm	NR	S,U	8 hr	LC50	10,000	Mills, 1977
	NR	NR, site 1	20	S,U	36 hr	LC50	10	Ferguson, et al., 1965a
	NR	NR, site 2	20	S,U	36 hr	LC50	30	Ferguson, et al., 1965a
	NR	NR, site 3	20	S,U	36 hr	LC50	25	Ferguson, et al., 1965a
	NR	NR, site 4	20	S,U	36 hr	LC50	<10	Ferguson, et al., 1965a
	NR	NR, site 5	20	S,U	36 hr	LC50	20	Ferguson, et al., 1965a
	NR	NR, site 6	20	S,U	36 hr	LC50	15	Ferguson, et al., 1965a
	NR	NR, site 7	20	S,U	36 hr	LC50	<200	Ferguson, et al., 1965a
	Tech.	Adult, site 1	21.1	S.U	36 hr	LC50	10	Boyd and Ferguson, 1964
	Tech.	Adult, site 2	21.1	S,U	36 hr	LC50	160	Boyd and Ferguson, 1964
	Tech.	Adult, site 3	21.1	S,U	36 hr	LC50	60	Boyd and Ferguson, 1964
	Tech.	Adult, site 4	21.1	S,U	36 hr	LC50	480	Boyd and Ferguson, 1964
	NR	NR, site 1	NR	S,U	48 hr	LC50	31	Dziuk and Plapp, 1983
	NR	NR, site 2	NR	S,U	48 hr	LC50	212	Dziuk and Plapp, 1973
	NR	NR, site 3	NR	S,U	48 hr	LC50	301	Dziuk and Plapp, 1973

TABLE 2.4.1: LABORATORY ACUTE MORTALITY DATA (Continued)

Organism	Toxicant Formulation	Life Stage or Size	Temperature (°C)	Test Method (Static, Flow-Through, Renewal; Measured, Unmeasured)	Exposure Duration[1]	Endpoint	Endpoint Concentration (µg/l)	Reference
Mosquitofish, *Gambusia affinis*	Floating	0.32 g	20 test water 1	S,U	96 hr	LC50	24	Workman and Neuhold, 1963
	Sinking	0.32 g	20 test water 1	S,U	96 hr	LC50	48	Workman and Neuhold, 1963
	Floating	0.32 g	20 test water 2	S,U	96 hr	LC50	52	Workman and Neuhold, 1963
	Sinking	0.32 g	20 test water 2	S,U	96 hr	LC50	6	Workman and Neuhold, 1963
	Floating	0.32 g	20 test water 2	S,U	96 hr	LC50	9	Workman and Neuhold, 1963
	Sinking	0.32 g	20 test water 3	S,U	96 hr	LC50	9	Workman and Neuhold, 1963
	NR	0.5 g	24	S,U	96 hr	LC50	10	Carter and Graves, 1972
	NR	30-40 mm	24	NR,U	96 hr	LC50	8	Chaiyarach, *et al.*, 1975
Striped bass, *Morone saxatilis*	Tech.	Juvenile; 2.3 g	17	F,U	96 hr	LC50	4.4	Korn and Earnest, 1974
Bluegill, *Lepomis macrochirus*	Tech.	NR	19.2	F,M	48 hr	LC50	2.3	Auwarter, 1977
	Tech.	NR	20.5	F,M	72 hr	LC50	1.5	Auwarter, 1977
	Tech.	0.6-1.5 g	12.7	S,U	96 hr	LC50	3.2	Macek, *et al.*, 1969
	Tech.	0.6-1.7 g	18	S,U	96 hr	LC50	18	Macek and McAllister, 1970
	Tech.	0.6-1.5 g	18.3	S,U	96 hr	LC50	2.6	Macek, *et al.*, 1969
	Tech.	0.5-1.5 g	20	S,U	96 hr	LC50	2.6	Macek, *et al.*, 1969
	Tech.	0.5-1.5 g	20	F,M	96 hr	LC50	4.7	Johnson and Julin, 1980
	Tech.	0.5-1.5 g	20	S,U	96 hr	LC50	2.4	Johnson and Julin, 1980
	NR	0.5 g	23	S,U	96 hr	LC50	4	Carter and Graves, 1972
	Tech.	0.6-1.5 g	23.8	S,U	96 hr	LC50	2.4	Macek, *et al.*, 1969
	Tech.	0.8 g	24	S,U	96 hr	LC50	2.4	Johnson and Finley, 1980
	Tech.	0.5-1.5 g	25	F,M	96 hr	LC50	3.4	Johnson and Julin, 1980
	20% a.i.	3.8-6.4 cm, 1.0-2.0 g	25	S,U	96 hr	LC50	3.5-4.6	Henderson, *et al.*, 1959
Largemouth bass, *Micropterus salmoides*	Tech.	0.9 g	18	S,U	96 hr	LC50	2	Johnson and Finley, 1980
Redear sunfish, *Lepomis microlophus*	Tech.	0.6-1.7 g	18	S,U	96 hr	LC50	13	Macek and McAllister, 1970
Yellow perch, *Perca flavescens*	Tech.	1.4 g	18	S,U	96 hr	LC50	12	Johnson and Finley, 1980

TABLE 2.4.1: LABORATORY ACUTE MORTALITY DATA (Continued)

Organism	Toxicant Formulation	Life Stage or Size	Temperature (°C)	Test Method (Static, Flow-Through, Renewal; Measured, Unmeasured)	Exposure Duration[1]	Endpoint	Endpoint Concentration (µg/l)	Reference
Bullfrog, Rana catesbeiana	Tech.	Larva	NR	F,M	96 hr	LC50	99	Hall and Swineford, 1981
Leopard frog, Rana sphenocephala	Tech.	Eggs	20	F,M	96 hr (20 days post exposure)	LC50	46	Hall and Swineford, 1980
	Tech.	Young larva	20	F,M	96 hr (26 days post exposure)	LC50	32	Hall and Swineford, 1980
	Tech.	Sub-adult	20	F,M.	96 hr (4 days post exposure)	LC50	378	Hall and Swineford, 1980
Wood frog, Rana sylvatica	Tech.	Larva	NR	F,M	96 hr	LC50	195	Hall and Swineford, 1981
American toad, Bufo americanus	Tech.	Larva	NR	F,M	96 hr	LC50	34	Hall and Swineford, 1981
Northern cricket frog, Acris crepitans	Tech.	Larva	NR	F,M	96 hr	LC50	76	Hall and Swineford, 1981
Western Chorus frog, Pseudacris triseriata triseriata	Tech.	Tadpole, 7 day	15.5	S,U	96 hr	LC50	500	Sanders, 1970
Fowler's toad, Bufo woodhousii fowleri	Tech.	Tadpole, 28-35 day	15.5	S,U	96 hr	LC50	140	Sanders, 1970
Spotted salamander, Ambystoma maculatum	Tech.	Larva	NR	F,M	96 hr	LC50	34	Hall and Swineford, 1981
Marbeled salamander, Ambystoma opacum	Tech.	Larva	NR	F,M	96 hr	LC50	342	Hall and Swineford, 1981

[1] The longest time for which endpoint values were calculated in each reference; all 96 hour values also presented.

[2] Information not recorded in publication.

[3] Emulsifiable concentrate formulation.

[4] Commercial grade toxaphene.

[5] Effect concentration for immobilization or death of 50% of test organisms.

[6] Technical grade toxaphene.

[7] Time to kill 50% of test organisms.

[8] Organisms collected from different field sites.

[9] Time to immobilize or kill 50% of test organisms.

One additional biological factor influencing vulnerability to toxaphene is the development of a resistance resulting from exposures which kill the more sensitive individuals in field populations. This phenomenon has been demonstrated for several fish and invertebrate species (Table 2.4.1) collected in areas of high agricultural use (Dziuk and Plapp, 1973; Albaugh, 1972; Naqvi and Ferguson, 1970; Klassen, et al., 1965; Ferguson, et al., 1965; Ferguson, 1968). Levels of resistance more than two orders of magnitude greater than for individuals from areas not contaminated by toxaphene have been detected in Missisippi Delta mosquito fish (Ferguson, 1968). The degree of resistance appears to correspond to the level of contamination and to be genetically rather than physiologically mediated (Ferguson, 1968). It is unlikely therefore, that exposure-related resistance is a significant factor in regard to the effects of toxaphene on aquatic organisms in the Great Lakes.

The acute sensitivities (up to 96 hours' exposure) of most fish species and the more sensitive invertebrates are all quite similar (Table 2.4.1). The lowest values range between 1 and 10 µg/L for both groups. The most vulnerable amphibians tested are slightly less sensitive (LC_{50}s ~ 30-35 µg/L) than fish, and the very limited algal data (Table 2.4.2) indicate considerably less sensitivity (EC_{50}'s 100 - 1000 µg/L). These laboratory data appear to relate well to the substantial body of information from the fish eradication-related studies cited previously. All fish species were found to be similarly sensitive in the field, but older fish were more resistent than young ones (Henegar, 1966). Treatment levels recommended for fast, complete eradication of fish (~10 to 200 µg/L depending on water quality) correspond to fish lethality levels observed in laboratory studies (Stringer and McMynn, 1960; Henegar, 1966; Webb, 1980; Hemphill, 1954; Kallman, et al., 1962; Needham, 1966; Woolitz, 1962; Cushing and Olive, 1956; Rose, 1958). Field results also agree with one another and with the laboratory data which show that many invertebrate species are less sensitive than fish; that some midges (especially Chaoborus sp.), amphipods, copepods, cladocerans, protozoans and odonates are among the most sensitive invertebrates (Hilsehoff, 1965); that oligochaetes, snails, leeches and many insects are quite resistent; and that plants and phytoplankton are resistant.

The subacute laboratory data (Table 2.4.2), although much more limited, indicate a sensivivity of about one to two orders of magnitude greater than the acute data for the same species. Effects were observed through the lowest exposure concentration (0.039 µg/L) in the brook trout partial life-cycle chronic test conducted by Mayer, et al. (1975). If one considers the similarity of acute values for most fish species (at least those tested for chronic toxicity), and if one assumes comparably close acute-chronic ratios (mean 96-hr LC_{50}: geometric mean of chronic effect and no-effect concentrations) for the fish in Table 2.4.2, brook trout are indicated to be only a little more sensitive than fathead minnows or

TABLE 2.4.2: SUBACUTE LABORATORY TEST RESULTS

Organism	Nature of Study	Toxicant Formulation	Temperature (°C)	Test Method Static Flowthrough, Renewal; Measured, Unmeasured	Effect Conc., or Effect/No Effect Conc. Range and Geom X (µg/L); Endpoint	Reference
			CHRONIC TOXICITY			
Green alga, Scenedesmus quadricauda	Exponential growth for 10 days	NR	21	S,U	100-1000; cell number	Stadnyk, et al., 1971
Cladoceran, Daphnia magna	Partial life cycle chronic test	Tech.	18	F,M	0.07-0.12, X=0.09; young production	Sanders, 1980
Amphipod Gammarus pseudolimnaeus	30-day growth test	Tech.	18	F,M	0.13 - 0.25, X=0.18; growth	Sanders, 1980
Midge (larva), Chironomus plumosus	30-day emergence test	Tech.	22	F,M	1.0 - 3.2	Sanders, 1980 X=1.8; emergence
Brook trout, Salvelinus fontinalis	Partial life chronic test	Tech.	9	F,M	0.039; fry growth and survival	Mayer and Mehrle, 1978
Fathead minnow, Pimephales promelas	Life cycle chronic test	Tech.	25	F,M	0.025-0.054, X=0.037; fry growth	Mayer, et al., 1975
Channel catfish, Ictalurus punctatus	Partial life cycle chronic test	Tech.	26	F,M	0.129-0.299 X=0.196; fry survival and growth	Mayer, et al., 1975
			PHYSIOLOGICAL, BEHAVIORAL, OTHER EFFECTS			
Brook trout, Salvelinus fontinalis	Partial life cycle chronic test	Tech.	9	F,M	0.039; vertebrae collagen metabolism	Mayer, et al., 1975
Goldfish, Carassius auratus	96-hr behavioral test series	NR	25	F,U	1.8; change from control	Warner, et al., 1966
Channel catfish, Ictalurus punctatus	Partial life cycle chronic test	Tech.	26	F,M	0.049-0.072, X=0.059; bone quality	Mayer, et al., 1977
Mosquitofish, Gambusia affinis	Behavioral Test	NR	NE	F,U	250; avoidance	Kynard, 1964
	8-hr intoxication test	NR	NR	S,U	12,000; EC50 equilibirum	Mills, 1977
	In vitro physiological test	NR	37	-	25; succinic dehydrogenase activity in brain & liver	Moffett and Yarbrough, 1972
Bluegill, Lepomis macrochirus	21-and 42-day exposures	Tech.	19.2-20.5	F,M	0.144; liver cytochrome P-450 level	Auwarter, 1977
Leopard frog, Rana spenocephala	96-hr intoxication test	Tech.	20	F,M	45; EC50	Hall and Swineford, 1980

channel catfish. Acute-chronic ratios for fathead minnows and channel catfish are similar (360 and 98, respectively) and perhaps even comparable when based on physiological effect endpoints. If one uses the highest of these ratios and the single available brook trout 96 hr LC_{50} concentration (10.8 µg/L, Table 2.4.1) to estimate a 'safe' chronic concentration for brook trout, a value of 0.03 µg/L is obtained, which is close to the 0.039 µg/L effect value observed in the chronic test. Sensitive invertebrates appear from chronic results to be more tolerant than fish, a condition which agrees with observations from the many fish eradication field studies that many invertebrate fish-food organisms survived treatments intended to remove fish.

Chronic toxicity data are probably most appropriate for evaluating the aquatic hazard of toxaphene because of its observed persistence in many water bodies. If it may be assumed that the 1 to 4 ng/L of toxaphene measured in Lakes Huron and Superior are near to being representative of concentrations in all the Great Lakes, then these lake levels are only about an order of magnitude lower than concentrations known to adversely affect sensitive aquatic organisms in long-term laboratory investigations. It would appear, therefore, that aquatic life may not be jeopardized by existing lake water concentrations, but with the proximity of these levels to known chronic effects, a great deal more quantitative information on lake water concentrations is urgently needed to better define this risk.

Bioconcentration data from chronic laboratory tests (Table 2.4.3) indicate that a water-to-tissue equilibrium of toxaphene concentrations is reached in fish at about 30 days of exposure. Pooling whole body residue data for all fish species provides a mean bioconcentration factor (BCF = tissue concentration : water concentration) of 23,667. Daphnia magna accumulated 4,000 times the water concentration of toxaphene. These values are similar to those observed by Terriere, et al. (1966) in several stocked fish species and other aquatic organisms from two Oregon lakes studied over a 3-year period during their recovery from fish eradication treatment. BCFs there ranged from 9,000 to 19,000 for rainbow trout, 4,000 to 8,000 for Atlantic salmon and 15,000 for brook trout. Residues in caged rainbow trout introduced into one of the lakes indicated an equilibrium might have been reached between 38 and 46 days of exposure. Invertebrate residues ranged between 1,200 and 2,500 times water concentrations and aquatic plants had BCFs of 500 to 7,000. The similarity of the laboratory (direct uptake) and field BCF data (within a factor of 3 or 4 for fish and invertebrates) indicates little or no food web magnification among entirely aquatic species. This phenomenon is different from the one discussed previously involving the mobilization of residues from contaminated sediments by benthic organisms or the accumulation in terrestrial organisms (e.g., wildfowl) from eating contaminated aquatic foods.

TABLE 2.4.3: LABORATORY BIOCONCENTRATION TEST RESULTS

Organism	Life Stage[1]	Toxicant Formulation	Exposure Duration	Test Method (Static Flowthrough, Renewal; Measured, Unmeasured)	Exposure Concentration Range	Tissue	Mean BCF[2,3]	Reference
Cladoceran, *Daphnia magna*	NR	Tech.	7 day	F,M	0.06-0.12	W.B.	4,000[3]	Sanders, 1980
Brook trout, *Salvelinus fontinalis*	Fry	Tech.	7 day	F,M	0.039-0.503	W.B.	14,000[4]	Mehrle and Mayer, 1975b
	Fry	Tech.	15 day	F,M	0.039-0.503	W.B.	65,000[4]	Mehrle and Mayer, 1975b
	Fry	Tech.	60 day	F,M	0.039-0.139	W.B.	12,000	Mehrle and Mayer, 1975b
	Fry	Tech.	90 day	F,M	0.039-0.139	W.B.	18,000	Mehrle and Mayer, 1975b
	Yearling	Tech.	161 day	F,M	0.039-0.502	W.B.	7,400	Mehrle and Mayer, 1975b
Fathead minnow, *Pimephales promelas*	Fry	Tech.	30 day	F,M	0.013-0.173	W.B.	23,000	Mehrle and Mayer, 1975b
	40 day	Tech.	30 day	F,M	0.013-0.173	W.B.	23,000	Mehrle and Mayer, 1975b
	40 day	Tech.	98 day	F,M	0.013-0.173	W.B.	17,000	Mehrle and Mayer, 1975b
	10 day	Tech.	150 day	F,M	0.055-0.621	W.B.	92,000[5]	Mehrle and Mayer, 1975b
	40 day	Tech.	295 day	F,M	0.013-0.173	W.B.	6,600	Mehrle and Mayer, 1975b
Channel catfish *Ictalurus punctatus*	Fry	Tech.	15 day	F,M	0.049-0.299	W.B.	9,100[5]	Mayer, *et al.*, 1977
	Fry	Tech.	30 day	F,M	0.049-0.299	W.B.	18,000	Mayer, *et al.*, 1977
	Adult	Tech.	50 day	F,M	0.049-0.299	W.B.	15,000	Mayer, *et al.*, 1977
	Fry	Tech.	60 day	F,M	0.049-0.299	W.B.	24,000	Mayer, *et al.*, 1977
	Adult	Tech.	75 day	F,M	0.049-0.299	W.B.	14,000	Mayer, *et al.*, 1977
	Fry	Tech.	75 day	F,M	0.049-0.299	W.B.	15,000	Mayer, *et al.*, 1977
	Fry	Tech.	90 day	F,M	0.049-0.299	W.B.	40,000	Mayer, *et al.*, 1977
Bluegill, *Lepomis macrochirus*	NR	Tech.	48 hr	F,M	1.41-3.36	W.B.	1,060[5]	Auwarter, 1977
	NR	Tech.	96 hr	F,M	0.71-1.49	W.B.	1,480[5]	Auwarter, 1977
	NR	Tech.	42 hr	F,M	0.036-0.144	Lipid	350,000[6]	Auwarter, 1977

Fish $\bar{X}$ BCF = 23,667; N = 15

[1] Life stage at which exposure to toxaphene started but excluding any embryonic exposure time

[2] BCF = tissue concentration of toxaphene ÷ water concentration of toxaphene

[3] BCFs at all concentrations for a given exposure circumstance (each row in the table) were nearly identical so table BCF values represent means for the 2 or more exposure concentrations

[4] High values assumed to be related to yolk retained by fry, so omitted from mean BCF calculation

[5] Probably not in equilibrium because exposure of less than 30 days and hence omitted from calculation of mean BCF

[6] No data provided by author to permit conversion to whole body values

Sublethal toxicity to mammals can be categorized in 4 basic types:

(a) General toxicity
A variety of studies on rats fed daily doses of toxaphene up to 1500 mg/kg for 7 to 10 days produced a number of functional and structural alterations of unknown significance (Villeneuve, in press). Less severe abnormalities were produced in dogs and monkeys. Lifetime feeding at rates of 25, 100 and 400 µg/g produced non-specific liver cell alterations such as those seen in shorter, subacute experiments, i.e., centrilobular hepatic cell swelling with increased oxyphelia and margination of basophil granules (Fitzhugh and Nelson, 1951). Toxaphene fed to rats at 25 and 100 µg/g and mice at 25 µg/g in chronic reproduction studies did not produce evidence of abnormalities in the mating index, fertility index, pregnancy index, parturation index, mean viable litter size, live birth index, lactation index or weanling body weights (Kennedy, et al., 1973; Keplinger, et al., 1970).

(b) Teratogenicity
Oral intubation of 15-35µg of toxaphene/g on days 7-16 of gestation produced encephaloceles in mouse offspring (Chernoff and Carver, 1976). Fetal mortality was increased in mice at all dosages and maternal mortality in rats and mice.

(c) Mutagenicity
Toxaphene did not induce dominant lethal gene mutations in mice (Epstein, et al., 1972). Mutagenicity is present in Salmonella test systems (Ames' test) without liver homogenate activation (Hooper, et al., 1979).

(d) Carcinogenicity
In chronic toxaphene feeding studies in mice and rats, abnormalities were observed at high doses (100 µg/g and higher)(NCI, 1979). Rats developed follicular cell carcinomas or adenoma of the thyroid. Hepatocellular carcinoma and liver cell adenomas were produced in mice.

Some accidental poisoning incidents have been described in humans; several of those involving children resulted in death (Villeneuve, in press). The major concerns remain those of occupational exposures in industry and agriculture (U.S. EPA, 1980). Individuals involved in the manufacture of toxaphene for periods of 2-18 years were reported as having no "adverse effects that could be associated with camphechlor (toxaphene) exposure" (Deichman, 1973). On the other hand, persons working in recently sprayed fields were found to have a variety of non-fatal signs and symptoms, i.e., dyspnea, decreased pulmonary function and "miliary" opacities on chest x-rays in one group and a variety of chromosome abnormalities, including acentric fragments and chromatid exchanges in another group (Embry, 1972). In a study no longer possible under current guidelines for the protection of human volunteers, no abnormalities were found in the

blood and urine of volunteers exposed to an aerosol of toxaphene in a closed chamber for 30 minutes a day on ten consecutive days (Villenueuve, in press). They also received the same exposure on three consecutive days after a 3-week period, and were estimated to have absorbed about 1 µg/g/day. No physical abnormalities were found in this acute/subacute study and no followup was performed.

There are few convincing environmental health data indicating toxaphene as a carcinogen, mutagen or teratogen in man. Two cases of acute aplastic anemia were recorded following an accidental lindane:toxaphene exposure (IARC, 1979). One of these persons died of acute myelomonocytic leukemia. An increased incidence of pulmonary cancer was reported in 285 agricultural workers applying various pesticides, among which was toxaphene (Barthel, 1976). Despite the paucity of evidence for human chronic toxic effects, the presence of carcinogenicity in rodents has prompted the U.S. EPA (1980) to propose human health criteria of 7.1 to 0.07 ng of toxaphene/L of water, corresponding to risk levels of 10^{-5} to 10^{-7}. These water concentrations are based on the human consumption of fish that bioconcentrate residues by a factor of 13,100 times in laboratory exposures.

Environmental Concentration Regulation

A variety of toxaphene water quality standards or ways of calculating such standards to protect humans or aquatic life and wildlife are specified in the pertinent regulations of the Great Lakes states. To protect aquatic life, several states propose the use of a Water Quality Criteria value of 0.01 µg/L (NAS/NAE, 1973); others specify multiplication of acute toxicity values* for resident species by a 0.1 or 0.01 application factor. New York requires a "nondetectable" level as defined by specified analytical procedures. Pennsylvania requires adherence to the existing 0.008 µg/L IJC objective. Several states also specify different toxaphene standard values or ways of deriving them for different water uses or classifications (e.g., in Illinois, 0.1 times the 96 hr LC_{50} for native fish for "general use" water, 5µg/L for drinking and food processing water). The Ontario Ministry of the Environment specifies a value of 0.008 µg/L related to the the protection of aquatic life and wildlife. The U.S. EPA published ambient water quality criteria for toxaphene in 1980 (U.S. EPA, 1980) and as of November 1, 1984, four states (Arkansas, Delaware, Maryland and Oklahoma) and the U.S. Trust Territories had adopted these freshwater aquatic life values -- not to exceed 0.013 µg/L as a 24-hour average and 1.6 µg/L at any time -- as legally enforceable water quality standards (U.S. EPA, personal communication).

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* U.S. National Academy of Science/National Academy of Engineering

To summarize the preceding discussion, a water concentration of about 0.006 µg/L would likely be non-toxic to the aquatic organisms and wildlife of the Great Lakes. Such a value is obtained by applying a 5-fold safety factor to the limited, sensitive organism chronic toxicity data of approximately 0.03 µg/L. On the other hand, Great Lakes water concentrations of 0.001 to 0.002 µg/L appear to have resulted in tissue residues up to 5 times the 5 µg/g FDA action level. Therefore, water concentration should be reduced by approximately 5-fold to 0.0002 µg/L to keep fish tissues below 5 µg/g. This concentration may be reduced further to protect humans, according to the assumptions employed by the U.S. EPA (1980) in calculating water concentrations associated with 10^{-5} to 10^{-7} cancer risk levels, if health data more soundly establishing toxaphene as a cancer causing agent in humans are generated.

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3. Report on Work Group on Indicators of Ecosystem Quality

3.1. Ecosystem objectives.

The AEOC is also investigating the use of biological measures or indicators of ecosystem health for the oligotrophic system in Lake Superior (and elsewhere) as well as other systems in other Lake basins.

The lake trout in Lake Superior and the ecosystem implications are fully discussed in a report entitled "A Conceptual Approach for the Application of Biological Indicators in the Ecosystem Basin". The report, which has received considerable external review, discusses the applicability of the indicator or surrogate concept within the context of the ecosystem approach. The report contains general criteria for the use of indicator species and the specific rationale for using the lake trout as an indicator species for oligotrophic Great Lakes' systems.

The Work Group's report provides a comprehensive overview of the critical elements known to control population dynamics of the lake trout. Such elements include: stocking/culture of lake trout and their competitors; sea lamprey control; influence of exotic species; habitat restoration and protection; and, of course, water quality management. In these traditional areas of determining the status of fish health and in others such as community interaction and biochemical indicators of stress, a need for more information and hence research options is noted. Thus, the report provides the background for recommending a general ecosystem objective and sets forth a methodology for assessing the achievement of a specific, cold water oligotrophic ecosystem objective.

In order to determine the utility of such a specific ecosystem objective for Lake Superior, the Work Group has developed a prototype computer program using the lake trout as the indicator species. This program is intended to assist fishery and water quality managers in integrating data and identifying stresses to ecosystem health.

The completion of this report and the development of the computer program do not conclude the effort on indicators, for, as set forth in the report, the use of other indicator species is clearly warranted. Work on a mesotrophic system in Lake Erie is planned.

The AEOC is satisfied with the feasibility and appropriateness of this approach and recommends that work be continued on the further development of biological indicators of ecosystem health, selecting species or communities for mesotrophic and eutrophic systems in addition to the lake trout for oligotrophic conditions.

4. Research and Other Data Needs

4.1 GENERAL NEEDS

4.1.1 Community Structure and Function

In order to develop alternative ecosystem indices for the health of different parts of the Great Lakes, it will be necessary to have a better understanding of the relationships among the various components of such sub-systems. Much of the necessary data may exist at present; other data may be needed. Multidisciplinary discussions should be fostered in order to determine precisely the additional data needs.

4.1.2 Lake Erie Fish Community Data

The fish community data base for Lake Erie should be examined prepatory to the development of a mesotrophic ecosystem objective. Additionally, data relating to organisms that interact and co-exist with the terminal predator in the lake should also be investigated. The AEOC intends to establish a work group to prepare a document utlizing such data, especially as they relate to the walleye.

4.1.3 Sediments as a Source of Toxic Chemicals

The adsorption of metals and toxic organic substances by sediments creates a major "sink" for many such substances. These can subsequently be released to the water and become available to biota--either directly as a toxicant or indirectly by accumulating through the food-web. Research is required to define and evaluate the transfer mechanisms including the conditions which control the rates and pathways of release. Sediment concentrations require identification as do threshold levels which cause adverse effects on appropriate biota. The toxic effects on many benthic species are also not well developed but should be used in objectives development.

4.1.4 Metal Speciation

Research to date has established that some metal species are more toxic to aquatic biota than others. Even though the use of 'total metal' is still perceived as a practical (and conservative) basis for water quality objectives, identification of the nature of these chemical species, including rates of their interconversions and development of suitable analytical methodology sensitive to 0.001-1 µg/L levels, is needed.

4.1.5 Air Sampling Methodology

While atmospheric deposition is established as an input mechanism for many of the persistent organic contaminants found in the Great Lakes, there are only partial data available on the concentrations and loadings via this route. Assessment of the significance of this pathway requires better sampling methods for vapour phase and particulate dry deposition.

4.1.6 Epidemiology

Epidemiological studies carried out by the U.S. EPA form the basis for the current recommendations for objectives on microbiological indicators and pathogens. This data base should be expanded to include Great Lakes conditions in beach areas, streams and small embayments. Efforts should also be expended to determine the relationships between historical indicators of microbial contamination and these newer, recommended objectives.

Additional work is also required to isolate, identify and enumerate the causative agents in cases where illnesses were associated with indicator organisms. Particular attention should be paid to the possibility that viruses may have been responsible for many of the illnesses described. Once routine analytical procedures for such agents have been established, they should be used in epidemiological studies.

4.1.7 Environmental Mapping

The description of a method for allocating limited use zones, as called for in the 1978 Agreement, will undoubtedly require the identification of sensitive inshore areas. Environmental mapping is recommended for this purpose and should include all sensitive uses (e.g. recreational swimming, spawning grounds, wetlands, etc.).

4.1.8 Quantitative Structure-Activity Relationships

Predictive relationships between the effects on aquatic organisms and the physical-chemical properties of chemicals are needed to fill the gaps in our data base for toxic chemicals found in the Great Lakes system. Research is needed to validate the existing equations and to develop new ones, especially for classes of compounds other than the phenols, anilines and halogenated benzenes.

4.1.9 Toxic Effects of Mixtures

Objectives developed to date, except that for Lake Superior Ecosystem Health (Lake Trout Indicator), have addressed the biological activity of single compounds. This procedure has largely been due to the lack of data dealing with interactions of among multiple contaminants. In the 1981 AEOC Report, a toxic unit approach was outlined for assessing the combined significance of metal mixtures found in the environment at less than objective levels. Research is needed to examine this approach and to correlate the toxic unit concept with effects found in the environment.

4.1.10 Mixing/Limited Use Zones

The 1978 Agreement and the IJC Second Biennial Report to the governments call for the determination of a mechanism for allocating limited-use zones of non-compliance. As work has not proceeded to satisfy this requirement attempts should be undertaken at least to describe methods for such allocations. These methods should take into account the interests of the Parties, the individual states and province, as well as the integrity of the system both at present and in the future.

4.1.11 Persistence

The approach to the development of objectives is often contingent on the definition of the persistence of a chemical. Hence, the conditions under which the concept is determined should be described. At present, persistence in any "natural environmental" system which demonstrates a half-life of eight weeks or more (or whose biota accumulate the substance) is accepted. A more precise definition is required and research is a precursor.

4.2 CHEMICAL SPECIFIC DATA NEEDS

The IJC's Co-ordinating Committee for the Assessment of Chemicals in the Great Lakes Ecosystem is proceeding with the task of describing procedure(s) for evaluating the hazards of chemicals in the environment. Previous AEOC reports have identified the following needs and it is anticipated that they will be required in any such future assessments.

4.2.1 Diazinon

Additional toxicity data are needed to assess bird mortalities resulting from the ingestion of organophosphate pesticide-treated crops or turf. Of particular interest is information on the extent to which successive exposures are cumulatively toxic and the extent to which the effects of different organophosphate pesticides are additive. In addition, life-cycle or long-term sublethal effects due to chronic exposures are needed for sensitive aquatic invertebrates such as insects and crustaceans.

4.2.2 Polynuclear Aromatic Hydrocarbons

Information exists to establish the carcinogenicity of some PAHs and/or their oxygenated metabolites. Other data show that aquatic organisms higher than the invertebrates metabolize the parent compounds readily. Two research-related activities are indicated:

- surveillance focussing on PAHs and their metabolites in appropriately polluted sub-systems in sediment, zooplankton, invertebrates (including insects and crustaceans) and fishes together with the establishment of levels of mixed function oxidases and the incidence of tumors are needed to establish the concentrations of the chemicals which may be responsible. This effort will require the development of appropriate methodology for the metabolites appropriate to the WHO recommended list -- fluoranthene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene and indeno(1,2,3-cd)pyrene and benzo(a)pyrene.

- determination of sublethal effects of chronic exposures on aquatic and benthic invertebrates. In this regard, the role of the release from the sediment of these materials will be important.

4.2.3 Polychlorinated Biphenyls, Heptachlor (including the epoxide) and DDT

The reliable determination of sub-nanogram/litre concentrations in filtered water has not been available to date. Present methodology should be capable of achieving this determination and should be applied to representative waters throughout the Great Lakes. The subsequent data, assuming detectable concentrations, are needed in conjunction with existing surveys for fish contaminants, to develop field values for the bioconcentration factors for these compounds.

4.2.4 Phthalates

Sublethal effects due to chronic, life-cycle exposure to phthalate esters by all types of aquatic freshwater organisms are needed. Some of this information exists for daphnids and midges exposed to dibutyl phthalate and di-2-ethylhexylphthalate, but for all other situations, it is unavailable. The effect of feeding at identified levels of contaminated foods for all organisms is also needed.

4.2.5 Organotins

Tributyl and trimethylin compounds have been found throughout the Great Lakes (especially nearshore areas and marinas) and the former is extensively used in the system. Sub-lethal effects on almost all freshwater organisms due to chronic exposures are needed.

4.2.6 Dieldrin

Sublethal effects on freshwater invertebrates arising from chronic exposures are needed.

4.2.7 Chlordane

Directly determined, no-effect or safe concentrations for trout species using sublethal responses as endpoints are needed. The impact of levels of chlordane in the diets of consumers of aquatic organisms is also required.

4.2.8 Mirex

Crayfish have showed delayed reaction (extensive mortality) to sub-microgram/litre levels of mirex. The no-effect level for crayfish from the Great Lakes and the responses of other crustaceans should be more precisely determined. Similar comments apply to photomirex. Indeed, the photoconversion of mirex--to photomirex and possibly kepone--should also be more clearly defined.

4.2.9 Silver

Scenedesmus spp. exhibit considerable toxic effects in the low µg/L range of silver. Other algae relevant to the Great Lakes should be tested to determine whether this phenomenon is general and, if so, at what levels.

4.2.10 Manganese, Molybdenum and Vanadium

Little or no sub-lethal effect data due to chronic exposures to these metals exist for fishes and similarly, neither lethal nor sub-lethal data exist for other aquatic organisms. Research is needed to fill this data gap.

4.2.11 Ammonia

Information is needed on the impact of reduced oxygen levels on ammonia sub-lethal toxicity to coldwater fishes at temperatures below 5°C. The influence of water hardness and alkalinity should also be determined.

Sublethal effects of ammonia on freshwater organisms other than fishes are a requirement, especially for aquatic invertebrates.

4.2.12 Chlorine

A routine analytical methodology effective at the 20 µg/L level and below for total residual chlorine is needed.

The effects of residual chlorine are inadequately known for photoplankton species in the Great Lakes. For fishes, lethality has been reported for some species (salmonines, fathead minnows) at or below the existing objective level but further information is required. Additional research is warranted to describe the impact on fish sublethal effects due to such chronic exposure to chlorine.

4.2.13 Oxygen

Fish mortality due to toxicants or other forms of stress at levels expected otherwise to be safe has been observed in oxygen depleted water. This phenomenon should be more extensively investigated for species likely to encounter such conditions (e.g. walleye in Lake Erie).

The rate at which most species acclimate to realistically reduced levels of oxygen (10%, 50% and 90% of saturation) is largely unknown. Knowledge of this rate would be extremely useful in determining the impact of lowered oxygen concentrations on such organisms.

4.2.14 Pentachlorophenol (PCP)

Uncertainty exists as to whether PCP is readily degraded in sediment-water systems relevant to the Great Lakes Basin. Also required is the rate and extent to which other substances (particularly the chlorobenzenes and chlorocyclohexanes) can form this chemical in the environment.

The sub-lethal effects of PCP on fishes are largely unknown although indications are that these occur at low µg/L levels. Effects have also been reported at such levels for one marine alga but the extent of this inhibition of freshwater algal species is unknown.

4.2.15 Dioxins

Photolysis rates of chlorinated dibenzodioxin congeners under conditions relevant to their occurrence in the Great Lakes is largely unknown. Studies should be undertaken using dioxins adsorbed to organic particulate matter suspended in water under both light and dark conditions.

Examinations of accumulators (sediments, predator fishes, gulls) should be undertaken for congeners other than 2,3,7,8-TCDD and particularly for the higher chlorinated ones.

4.2.16 Lead

In the Great Lakes, not enough is known quantitatively about the atmospheric input and an adequate budget for lead is difficult to prepare without this information.

Data exist on the sublethal effects due to the chronic exposure of freshwater biota to inorganic lead but the same is not true for alkylated lead compounds. The forms and availability of this metal in the aquatic system (see also 4.1.4 Metal Speciation) are also relevant to these toxic effects as well as to any system budget for this element.

4.2.17 Chromium

It appears that algae are among the most sensitive aquatic organisms to this chemical and that effects may be obtained at levels near those specified by the existing objective. Research is needed to establish such effects for a range of freshwater algal species in the Great Lakes.

4.2.18 Mercury

Mercury is found in fishes primarily in its methylated mercury form and it has been established that the methylation occurs in the sediments. Investigations are required to establish how and at what rates the mercury moves from the sediment into the fish.

4.2.19 Polychlorinated Styrenes

This group of chemicals has been detected in fish tissues from several of the Great Lakes and their structure is similiar to PCBs; but little else is known about them. Evaluation of their environmental significance requires data on emissions, possible atmospheric transport, sublethal effects due to chronic exposures of aquatic biota, persistence and mutagenicity.

4.2.20 Asbestos

A vast body of information exists on the effects of asbestos inhaled by mammals but little information is available on the effects of ingested material for aquatic organisms. Studies are underway on some of these concerns but emphasis is placed here on data concerning the interconversion of fibre types in mammalian systems and on effects on mammals which arise from chronic uptake in the gastrointestinal tract.

4.2.21 Guthion and Malathion

These pesticides exhibit lethality towards crustaceans and insects at levels which were the lowest observed for relevant Great Lakes aquatic biota. As a consequence, objectives were set using a procedure (0.05 x 96h-LC_{50}) for guthion and an arbitrary safety factor (0.2) applied to a threshold mortality for malathion. It would be preferable to employ sublethal effect data from chronic exposures for these organisms as the basis for the objectives.

4.2.22 Toxaphene

Since some fishes have been shown to be sensitive (acute lethality) to toxaphene, further investigations should be undertaken to determine the chronic toxicity to additional fish species. Chronic effects data are also needed for sensitive aquatic invertebrates. Studies on toxaphene carcinogenicity are also needed to reduce uncertainties that exist about the compound.

The presence of toxaphene in the Great Lakes has been well-established but the input sources have not been identified. Evaluations are needed on whether the current restrictions in Canada and the United States have been effective in limiting its introduction to the system. New data are needed in order to establish trends in bioaccumulation.

5. Future Directions

5.1 Chlorobenzenes and Chlorophenols

These compounds have been found extensively in different parts of the Great Lakes ecosystem and are known to have toxic effects on a variety of organisms. There is an existing objective for pentachlorophenol. Work will commence to determine whether it is possible to "fill-the-blanks" in the toxicology and other properties of these two classes of compounds by using structure-activity relationships and so to develop a "class" objective based on the relationships.

5.2 Assessment of Chemicals

Approximately 1000 compounds have been reported as being found in different parts of the Great Lakes ecosystem. The AEOC is participating, along with other IJC committees, in the work of the Co-ordinating Committee for the Assessment of Toxic Chemicals in the Great Lakes Ecosystem. The purpose is to provide a rational and consistent approach to determining the hazard of such compounds and to use this approach to establish priorities in developing future objectives and recommendations for research.

5.3 Mesotrophic Ecosystem Objective for Lake Erie

Parts of Lake Erie appear to be most appropriately described as a mesotrophic ecosystem. A work group will be established to prepare a report on an objective for a healthy mesotrophic system, based upon a native top predator as the suitable indicator species.

Appendix

TERMS OF REFERENCE

COMMITTEE MEMBERSHIP

ACKNOWLEDGEMENTS

TERMS OF REFERENCE FOR THE
AQUATIC ECOSYSTEM OBJECTIVES COMMITTEE OF THE
SCIENCE ADVISORY BOARD

The Aquatic Ecosystem Objectives Committee (AEOC) of the Science Advisory Board will:

1. Develop aquatic ecosystem objectives. Where feasible, these should be in the form of use effect curves, for various uses, and always including the most sensitive use.

2. Regularly review objectives and recommend amendment or introduction, based upon all available criteria.

3. Establish task forces to develop position papers on which to base the development of new or altered objectives.

4. Set general guidelines under which the objectives will be developed and define some minimum levels of scientific information at which an objective can be defined.

5. Develop an approach for the selection and ordering of parameters to be addressed.

6. Identify gaps in the knowledge needed to develop objectives and recommend the research required to fill the gaps.

MEMBERSHIP

The AEOC will consist of eight members: two aquatic toxicologists, three water quality specialists (one each from the provincial, state, and one of the federal governments), a limnologist, an aquatic chemist, and a human health aspects expert.

SUBMISSION OF PROPOSED OBJECTIVES

"Since the Science Advisory Board has the responsibility for advising on policy implications of proposed objectives on an ad hoc basis, the Commission plans to advise the Science Advisory Board to take the initiative in the study of new or revised water quality objectives, in consultation with the Water Quality Board as required, and to forward reports simultaneously to the Commission and the Water Quality Board. Thus, the study of objectives will not be dependent on actions of the Water Quality Board, but there will be an opportunity for the Board to advise the Commission on the practicability of the objectives under consideration or on the need for additional study from the Water Quality Board perspective". (Excerpt from a letter dated May 13, 1980, from the International Joint Commission to the Secretary of the Water Quality Board.)

REVISED AND APPROVED BY THE
SCIENCE ADVISORY BOARD
SEPTEMBER 3, 1980.

GREAT LAKES SCIENCE ADVISORY BOARD
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